VOLUME 10 *Heart Number* NUMBER 6

THE MEDICAL CLINICS OF NORTH AMERICA

MAY, 1927

Index Number

PHILADELPHIA AND LONDON
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THE MEDICAL CLINICS
OF
NORTH AMERICA

Volume 10 No. 6

INTRODUCTION

DAVID RIESMAN

IF I may repeat a story I have already told on myself, I will begin by saying that a number of years ago a friend and I while walking home over Walnut Street Bridge were discussing what we might do to add to the knowledge of an expectant world. After considering various subjects, we took up the physical diagnosis of diseases of the heart. That appealed to us; but neither of us could see any hope of adding anything of importance in this field, and we concluded that inasmuch as additions were impossible we had best devote our untapped energies to something else. Alas, we were "too young to know that we were young." Our prophecy was as bad as that of the great French pathologist, Andral, who in the year 1829 wrote the following paragraph:

"Since the publication of the immortal researches of Corvisart and of numerous other works subsequent to his, which have still further enlarged the dominion of science, the history of diseases of the heart and its membranes is to be regarded as almost complete."

Little did we know that a keen-witted Scot—a family practitioner in a small English town—was at that very moment laying the foundations for the new science of cardiology which has revolutionized our concepts regarding cardiac function, especially regarding the cardiac arhythmias. James Mackenzie

The contributions to this number of the Clinics represent the Seminar on Diseases of the Heart given under the auspices of the Philadelphia County Medical Society during the fall and winter of 1926–27.

met with great opposition and very naturally most of it came from the English pundits, who would not admit that any wisdom could come from a country doctor. As Mackenzie himself says, there are three stages in the history of every medical discovery: When it is first advanced people say it is not true; then a little later, when its truth has been borne in upon them so that it can no longer be denied, they say it is not important; after that, if its importance becomes sufficiently obvious, they say, "Anyhow, it is not new." Americans and Germans made pilgrimages to Burnley to sit at the feet of the Master. Later, Mackenzie moved to London, but his reception was cool; in the end, however, he triumphed and became the founder of the great English School of Cardiology—the world's present Mecca.

The new science is to a considerable extent based upon instrumental methods of research. Valuable as it is—and its value will be explained to you in the seminars that are to follow—it does not replace and can never replace or displace the old and tried procedures of physical diagnosis, based upon sight, hearing, and touch.

In planning the Cardiologic Seminars the Committee has had two main objects in view:

1. To restate in terms of practical experience the facts concerning the circulation that every medical man—physician, surgeon, and specialist—should know.

2. To lay before you the most recent discoveries in physiology, anatomy, and technic.

As will be seen from the program of lectures, a few will deal with the newer chapters in cardiology written by Mackenize and his school and by Einthoven and his disciples throughout the world. It was felt that since the present-day literature was so full of these subjects, an explanation of the fundamental principles by highly trained students would be welcomed by all. Not only should this information prove of interest in the abstract, but it ought to be helpful in enabling the general practitioner to make proper use, with a full realization of their limitations, of the electrocardiograph and other modern methods of precision.

Do not fear, however, that too much emphasis will be laid upon the recently acquired refinements or that the seminar will be conducted solely in the new cardiologic language—the speakers will talk in the vernacular.

The mortality statistics of all large cities show a disquieting increase in the deaths from heart disease, or diseases of the organs of circulation. It is reasonable to suppose that medical science can do a good deal to lessen the incidence of these diseases, but it will be possible only if there is a better understanding of their causes and of the means of their early detection.

I am indulging in the fond hope that in a few years from now we may see the results of this and other seminars in a reduction of the death rate from diseases of the heart.

THE CLINICAL VALUE OF THE ELECTROCARDIOGRAPH

WILLIAM D. STROUD

THIS article is presented primarily for the information of the general practitioner and with the hope that it may help to remove the fear of the average physician that any article which includes electrocardiograms must be too scientific to be of practical value. It contains few original opinions, and the author has availed himself freely of the writings of those best able to give a conservative estimate of the clinical value of the electrocardiograph.

Many physicians have been disappointed in not receiving from electrocardiograms the assistance toward an accurate diagnosis and prognosis in heart cases which they have been led to expect. This to no small degree is the fault of cardiologists who in their enthusiasm have over-emphasized the importance and results obtainable through electrocardiography. Such tracings are not the last word by any means in the diagnosis, prognosis, and treatment of patients with heart disease. Nothing at present can take the place of an accurate history and careful physical examination in estimating the myocardial reserve of each patient's heart. The electrocardiograph will help just as the *x*-ray helps in the diagnosis and prognosis of pulmonary tuberculosis, but *beware of the so-called cardiologist who attempts to make a clinical diagnosis from electrocardiograms alone.* Any technician can be taught to make pretty pictures, but it takes time and study to learn their correct interpretation and years of experience to properly evaluate their place in the clinical picture. Remember that just as a heart which clinically may appear normal may give an abnormal electrocardiogram, so also a heart with all the clinical signs of extensive pathologic damage may

give so far as our present knowledge has progressed, an apparently normal electrocardiogram.

Unfortunately, the average physician still depends upon the character and intensity of heart murmurs to make his diagnosis and prognosis and plans his treatment accordingly. Fifteen years' teaching has not yet been successful in proving to general practitioners that murmurs mean relatively little in the estimate of the all-important myocardial reserve. They should and do call attention to the heart, but we must all realize that an individual may have the loudest of murmurs and yet have sufficient myocardial reserve to prevent any circulatory embarrassment for many, many years.

Hence, the crucial point in the diagnosis, prognosis, and treatment of patients with heart disease is an estimate of their cardiovascular reserve. We must admit that this is a very difficult problem and that we have far to go before it will be in any way an easy matter. For many years a careful history and a thorough physical examination have been all that the physician has had upon which to base this estimate. The *x*-ray has been rather disappointing, although fluoroscopy has contributed something. The routine determination of the vital capacity has also been disappointing, so that it is natural for the physician to turn with eager anticipation to the electrocardiograph for help in this extremely difficult matter of estimating the cardiovascular reserve.

To attempt to explain the interpretation of electrocardiograms in such a brief article would only tend to add to the confusion in the mind of the average physician as to just what these pictures represent. If, however, any physician is willing to give six weeks of his time to attending one of the courses in cardiology continually in progress in various parts of the country, he will return to his practice with a thorough knowledge of electrocardiography and much better equipped to make an accurate clinical diagnosis and give satisfactory treatment to the average heart case, even though it may be impossible for him to have electrocardiograms made upon his patients.

In 1855, Kölliker and Müller[1] first demonstrated in the heart

of a frog that an electrical current was associated with each of the contractions. In 1887, Waller[2] made the first use of the capillary electrometer on human beings. In 1903, Einthoven[3] first introduced the string galvanometer, but it has been through the pioneer efforts of Sir Thomas Lewis[4, 5, 6] and his writings that the knowledge of the clinical value of the electrocardiograph has spread throughout the world. Figure 174 is a reproduction from page 21 of his large work on the Mechanism and Graphic Registration of the Heart Beat and warrants careful study as it well represents just what is going on in the heart during the tracing of an electrocardiogram. (See the line marked "ELECT." The letters "P," "Q," "R," "S," "T," "U" are merely six consecutive letters taken from the alphabet to indicate the various curves of an electrocardiogram and have no meaning in themselves.) A simple and brief description of just what electrocardiograms represent as produced through the string galvanometer is given by Lamson[7]: "In the electrocardiograph we have an instrument which has nothing to do with the actual contraction of the heart, but records the impulses for these contractions. The principle is a simple one, although the instrument is of formidable appearance. It is based on the fact that for a muscle to contract a stimulus is necessary and when the stimulus arises, whether it be of nervous, mechanical, or chemical nature, an electrical difference of potential is set up in the muscle just before its contraction. Such a disturbance occurs when any muscle contracts, and as the heart is a large muscle it causes a relatively large disturbance. These changes of potential can easily be recorded by placing the patient's hands in jars of salt solution connected by wires to the electrocardiograph. This is simply a very sensitive galvanometer or instrument for measuring small electral changes and an apparatus for recording them photographically. The simplest form of galvanometer is one in which a coil of wires is placed about a small compass. When there is a flow of electricity through the coil, the needle of the compass moves out of its normal position and when the current ceases the needle swings back again. . . . In the electrocardiograph an Einthoven galvanometer is used which

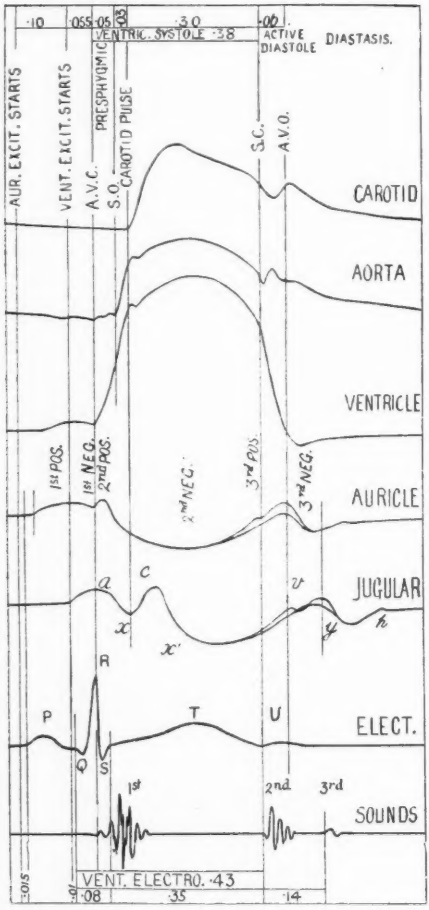

Fig. 174.—"A diagram intended to represent the curves of pressure in the carotid, aorta, left ventricle, and right auricle of the human subject; showing the supposed or ascertained time relations of these curves, the jugular pulse curve, the electrocardiogram, and apical heart sounds to each other. The diagram has been constructed so far as possible from curves taken from man, but where such are non-existent the diagram has been completed by curves obtained from the dog. . . . The times at which the excitation wave begins in auricle and ventricle are represented by vertical lines; the movements of the valves are also represented in a similar way. The scale

is made on a different principle from the compass galvometer. Here the movable part is a microscopic thread of quartz called a 'string,' which is suspended vertically in a strong magnetic field. When a minute current is passed through it the 'string' is deflected or really bent laterally. As the 'string' is supported at both ends, has a very small mass, and moves only a fraction of a millimeter, it has very little inertia and can record impulses up to many hundred times per minute. These records are obtained by making the string opaque with a coating of silver, placing it in a beam of light, which throws a vertical shadow, magnified by a microscope, on to a metal plate in which there is a horizontal slot. This slot allows only a point of shadow to pass through to a moving photographic plate or film on which the point of shadow writes in a continuous curve."

The apparatus as reproduced from Dr. Lamson's work is shown diagrammatically in Fig. 175, with a normal electrocardiogram beneath it (Fig. 176).

Although electrocardiographic tracings obtained upon specific cases are of much value; generally speaking, electrocardiography has been of just as much benefit to humanity in enabling the physician who is willing to devote the time to learning just what electrocardiograms represent, to more accurately interpret the physiologic and pathologic changes in the hearts of his patients. Although he may have previously known his patients well clinically, he will gradually find himself taking a renewed interest in an accurate diagnosis of the various arhythmias. He will find himself visualizing in his mind's eye just what is going on in that heart as it labors to do its work enclosed in the thorax, and as a result he will make more accurate diagnoses and prognoses and his patients will respond better and more as he expects them to respond to various forms of treatment. Finally, instead

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of abscissæ is 1 mm. = 0.015 sec. *A.V.C.* and *A.V.O.* = closure and opening of auriculoventricular valves, respectively; *SO.* and *S.C.* = opening and closure of semilunar valves, respectively. Other verticals are drawn at convenient points and the chief time intervals are marked above and below in seconds." (From Lewis, The Mechanism and Graphic Registration of the Heart Beat, Shaw and Sons, Ltd., London, Publishers.)

of dividing all heart cases into two classes: one which needs rest and digitalis, and one which does not, he finds each heart case a different and exceedingly interesting problem.

If a galvanometer is available with someone to interpret the tracings for us, or if we are able to make such interpretations

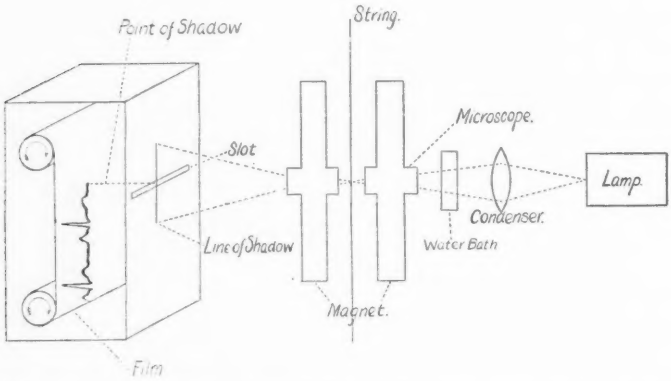

Fig. 175.—Diagrammatic representation of the electrocardiograph. (From Lamson, The Heart Rhythms, Williams & Wilkins Co., Publishers.)

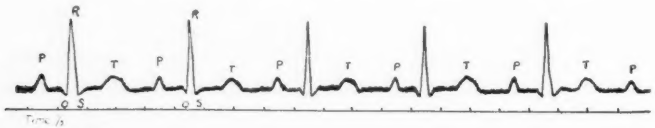

Fig. 176.—A diagrammatic electrocardiogram of a normal rhythm. The auricular impulse is shown by the upright curve *P*, and the ventricular impulse by the complicated curve or "complex" *QRST*, the beginning and end of which mark the beginning and end of systole. (From Lamson, The Heart Rhythms, Williams & Wilkins Co., Publishers.)

for ourselves, upon what type of patient will these electrocardiographic tracings prove most valuable?

First of all, in the cases of suspected arhythmias; namely, sinus arhythmia; premature contractions (extrasystoles); auricular fibrillation; auricular flutter and the various types of incomplete and complete heart-block, or delayed conduction.

Today every clinician if he is unable to accurately diagnose an arhythmia at the bedside should feel it his duty to have that arhythmia diagnosed by a consultant who has learned to be able to make such a diagnosis through his experience with electrocardiograms; or, if a galvanometer is available, to have the arhythmia diagnosed through electrocardiographic tracings. It is not sufficient to merely know that the heart is slightly irregular, or markedly irregular in its rhythm, for upon an accurate diagnosis of the type of arhythmia depends the treatment and the prognosis.

The second type of case in which electrocardiographic tracings are of definite aid is in a suspected enlargement of the heart, for it is upon this diagnosis that much dependency must be placed in estimating the amount of myocardial reserve present. If, clinically, we have border-line evidence of cardiac enlargement, an electrocardiogram giving definite evidence of a preponderance of either ventricle is certainly confirmatory evidence, and may mean much in the future treatment of the case.

Cardiac damage is seldom the cause of precordial pain in patients under the age of thirty-five except in the presence of very definite cardiac pathology which is demonstrable clinically. In patients beyond this age, however, we must be very careful in ignoring the heart as the cause of precordial pain even though we can demonstrate nothing abnormal clinically. It is here that electrocardiograms showing delay in conduction or definite bundle branch block or some preponderance of the ventricles may be the determining factor in placing the blame upon the heart. The work of Willius[8] is being accepted and confirmed by many cardiologists so that we are more and more realizing the importance of negativity of the T-wave in leads I and II, as indicative of a guarded prognosis, especially in patients complaining of precordial pain.

The galvanometer has done much to call our attention to the occurrence of coronary thrombosis. This diagnosis until recently was rarely made; but in the presence of those physical signs so well described by Wearn[9] we now know that occlusion of the coronary vessels is relatively common.

Finally, in children and young adults, in whom rheumatic activity in the heart is suspected, electrocardiograms indicating prolongation of auriculoventricular conduction often give confirmatory evidence; although, unfortunately, the absence of such a delay in conduction does not justify one in the thought that rheumatic activity is necessarily either over or latent.

Summary.—Thus, we find electrocardiograms help the physician by developing in him through the studies necessary to understand their interpretation a better understanding as to the physiologic and pathologic changes in the heart. Secondly, in patients in whom the diagnosis of heart disease is questionable, electrocardiographic interpretations will often confirm or throw doubt upon our clinical opinions and thus supply much assistance in estimating the myocardial reserve of each individual patient, which is, after all, the main information we need in order to make an accurate diagnosis and prognosis and to properly plan our treatment.

Note.—Following this lecture, Dr. John A. Sweeney demonstrated the technic of taking electrocardiograms.

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THE BUNDLE OF HIS AND ITS RELATION TO DISEASES OF THE HEART

JOHN EIMAN

THE causes and pathway of the heart-beat have interested physiologists and clinicians for centuries. Before William Harvey (1628) physicians followed the teachings of the old masters, Hippocrates, Aristotle, and Galen. They believed that in diastole the heart dilated actively and that this was due to implanted heat or vital spirits. Harvey proved that the acting movement of the heart was a contraction during systole, and that the contraction forced blood from the ventricles into the arteries. He did not speculate concerning the causes of the heart-beat, but he taught that the systole was an active contraction of the heart's own musculature and that this did not depend on external influences.

The discovery of nerve-cells in the frog's heart by Ramack (1848) stimulated investigation of this problem and led to the formulation of the *neurogenic theory* of the heart-beat by Volkmann. This theory assumed that excitation of each heart-beat arose in the nerve-cells situated at the junction of the large veins and the auricles, and that the stimuli generated within these nerve-cells were transmitted through their axones to the auricles and then to the ventricles. This theory assumed the existence in the heart of a principal motor center and dependent subordinate centers. This intrinsic nervous system was supposed to be responsible not only for the spontaneous origin of the heart-beat but also for its sequence and co-ordination.

Painstaking anatomic and physiologic studies, however, failed to support the above theory. Gaskell presented strong evidence that the nerve-cells in the heart belonged to the inhibitory mechanism of the heart. Gaskell, Engelmann, and

others elaborated the *myogenic theory* of the heart-beat. This theory assumed that the heart-muscle itself possessed the property of automatic rhythmicity and that this property was most highly developed in the sino-auricular node, situated near the

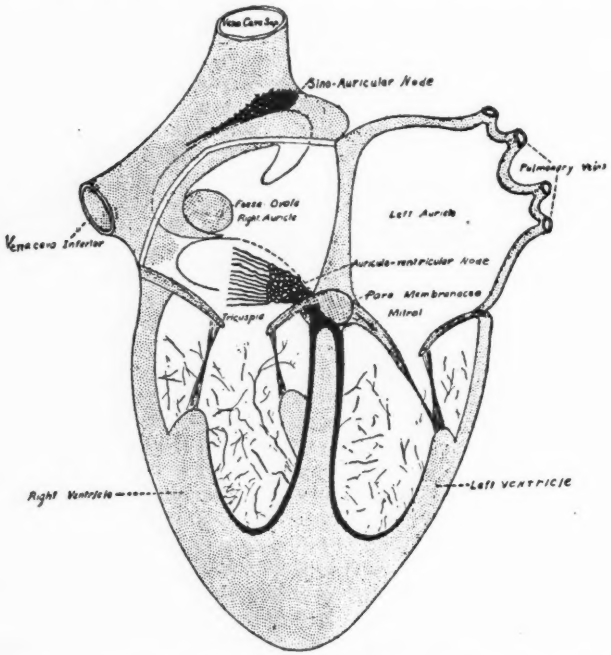

Fig. 177.—Diagram of specialized tissue. Normally the heart-beat originates in the sino-auricular node. From there the impulse spreads over the musculature of the auricle to the auriculoventricular node. There the impulse is conducted over the auriculoventricular bundle and its branches to the delicate arborizations of Purkinje's fibers and transmitted to muscle of the ventricles. (After Koch, Zeitschr. f. exper. Path. u. Therap.)

junction of the superior vena cava and the right auricle. By ingenious methods, Thomas Lewis has shown that the excitation wave spread from the sino-auricular node over the musculature of the auricles to the auriculoventricular node and from there over the auriculoventricular bundle, which, after crossing the

auriculoventricular groove, conveyed the impulse to the musculature of the ventricles through the left and right branches of the bundle and the network of Purkinje's fibers. We know now that the auricles and ventricles are connected by the auriculoventricular bundle and that the destruction of this bundle leads to heart-block. On the other hand, cutting of all detectable nerves crossing the auriculoventricular groove fails to alter the

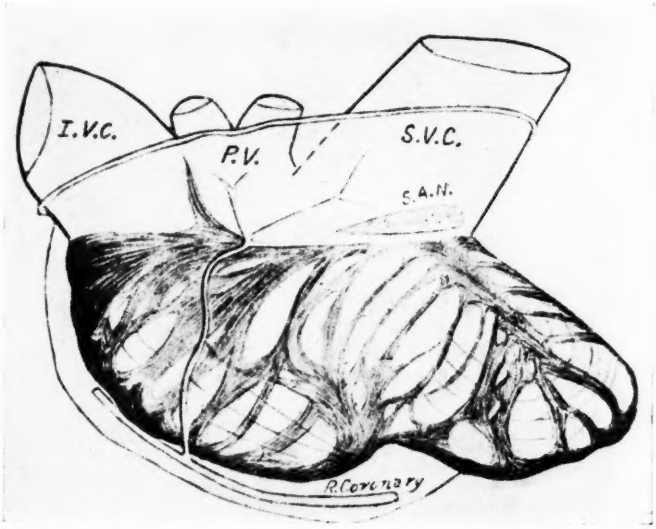

Fig. 178.—A drawing to scale of the right auricle of a dog showing the position of the sino-auricular node. *S.V.C.* and *I.V.C.* = superior and inferior venæ cavæ. *P.V.* = right pulmonary vein. *S.A.N.* = sino-auricular node. (After Thomas Lewis, The Mech. and Graph. Registr. of the Heart-Beat.)

rhythm and sequence of the heart-beat. At the present time the myogenic theory has been accepted by practically all physiologists and clinicians.

The sino-auricular node (Keith-Flack node) is situated at the sulcus terminalis near the junction of the superior vena cava and the right auricle. The node is about 20 mm. long and 2 mm. thick. It surrounds a small branch of the right circumflex artery.

It is composed of modified heart muscle-fibers, nerve endings, a few ganglia, and connective tissue. The muscle-fibers are small, about one-third the size of the heart muscle-fibers of the auricle, and their striations are indistinct. Keith and Flack have shown by dissection that the nerve-cells and nerve-fibers connect with vagal and sympathetic nerve trunks.

The auriculoventricular node (node of Tawara) is situated in the posterior right border of the inter-auricular septum. It is composed of small modified heart muscle-fibers. They are spindle

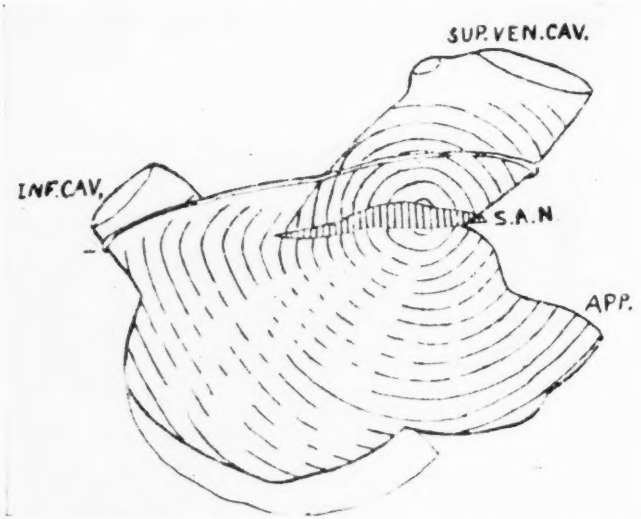

Fig. 179.—A diagram to illustrate the manner in which the excitation wave advances over the surface of the right auricle. The spread is from the upper part of the node and follows the chief muscle bands at almost uniform rates. (After Thomas Lewis, The Mech. and Graph. Registr. of the Heart-Beat.)

shaped and slender. Their striations are rather poorly marked and their bundles are irregularly arranged and interlaced with each other. The node shows numerous ganglion-cells and nerve-fibrils. The modified muscle-fibers, nerve-fibrils, and ganglion-cells are interwoven with connective-tissue cells. The ganglion-cells and

nerves connect with the vagus and sympathetic nerves. The node has a rich blood-supply, usually from the right circumflex artery.

The auriculoventricular bundle (bundle of His) begins in the auriculoventricular node. The bundle runs almost horizontally forward and to the left, then downward. It crosses the auriculoventricular groove and establishes a connection between the auricles and ventricles. Then the bundle divides into its right

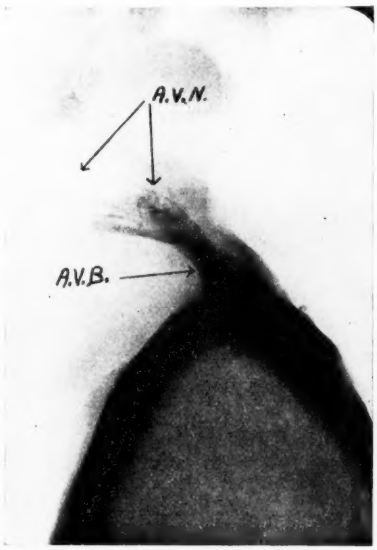

Fig. 180.—*x*-Ray of auriculoventricular node (*A.V.N.*), auriculoventricular bundle (*A.V.B.*), and its main branches after injection with potassium iodid solution.

and left branches. The left division enters the cavity of the left ventricle near the anterior limits of the membranous portion of the interventricular septum underneath the junction of the right anterior and posterior cusps of the aortic valves. The right branch enters the cavity of the right ventricle slightly in front of the anterior attachment of the septal segment of the tricuspid valve. The bundle and its branches are enclosed in a delicate

connective-tissue sheath, which is continuous with the sheath enclosing the divisions and the delicate arborizations of the branch bundles. The sheath appears to be continuous or encloses the auriculoventricular node (Fig. 180). The branch bundles and their division are situated directly beneath the endocardium.

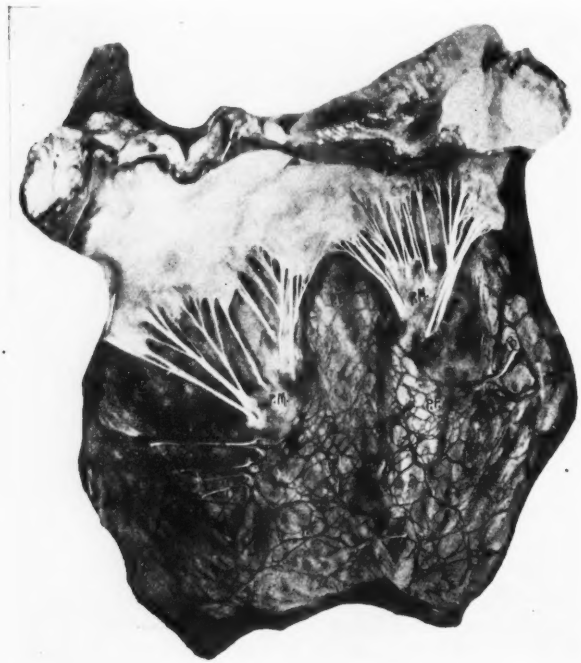

Fig. 181.—Photograph of branches (*B*) of the left branch bundle and the network of Purkinje's fibers (*P.F.*) in a beef heart after injection with india ink. *P.M.* = papillary muscles.

Histologically, the fibers of the auriculoventricular bundle and its branches vary with their situation, although all of them are modified heart muscle-fibers. The fibers of the main bundle are larger than the fibers in the nodes, still they are much smaller than normal heart muscle-fibers, their striations are fairly distinct and their nuclei are small and compact. Their arrangement

is roughly parallel. In the branch bundles and their divisions the fibers are large and tortuous, the protoplasm is less dense, and their nuclei are large and vesicular.

In the ventricles the branch bundles break up into smaller divisions and they, in turn, break up into very delicate arborizations of Purkinje's fibers. The network of these fibers envelops

Fig. 182.—Photograph of the right branch bundle (*R.B.B.*) and its arborizations after injection with india ink. *P.M.* = papillary muscles. *M.B.* = moderator band over which is seen large branch of the right bundle.

completely the cavities of the ventricles except the tips of the papillary muscles. The Purkinje's fibers communicate with the ventricular muscle-fibers. Fairly large, completely isolated branches of the left branch bundle surrounded by endocardium go directly to the papillary muscles in order to enable the stimuli to reach these structures without the slightest delay (Fig. 181, *B*).

These isolated branches have often been mistaken for moderator bands.

It is important to remember that the branch bundles, their subdivisions, and the network of Purkinje's fibers are located in

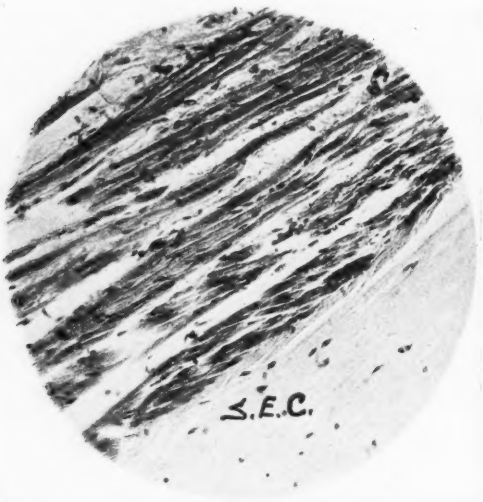

Fig. 183.—Section of auriculoventricular bundle showing small modified heart muscle-fibers. *S.E.C.*, subendocardial connective tissue.

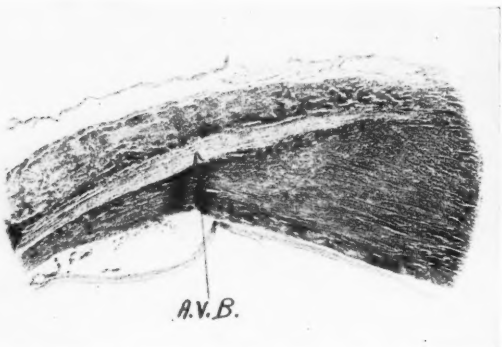

Fig. 184.—Auriculoventricular bundle piercing auriculoventricular groove.

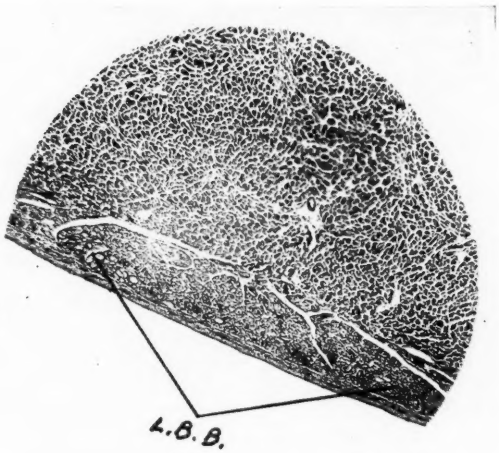

Fig. 185.—Cross-section of the left branch bundle (*L.B.B.*), low power.

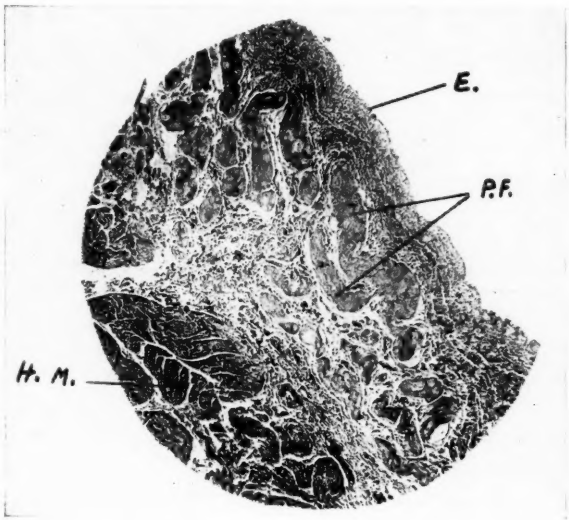

Fig. 186.—Section of a branch of the left branch bundle showing the situation of the Purkinje's fibers. *E* = endocardium and subendocardial connective tissue. *P.F.* = Purkinje's fibers. *H.M.* = heart-muscle.

the subendocardium. Consequently, any pathologic conditions affecting the subendocardium may destroy portions of the Purkinje's system and so interfere with the normal pathway of the cardiac impulse and lead to disturbance of the normal sequence of events in the cardiac cycle.

The outlining of the branch bundles by india ink or mercury injections is made possible by the fact that the auriculoventricular node, auriculoventricular bundle, its branches, and the delicate

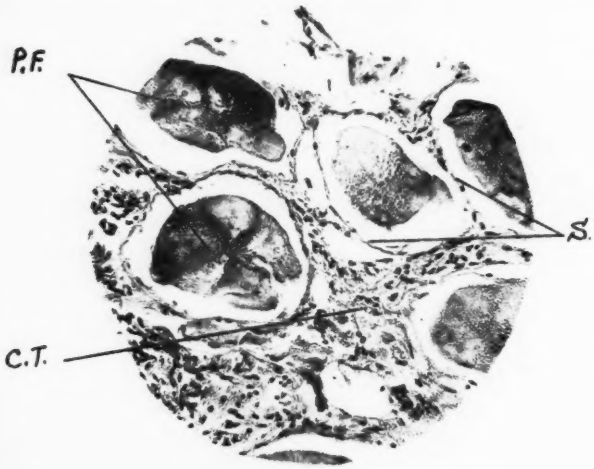

Fig. 187.—Section showing Purkinje's fibers (*P.F.*) surrounded by delicate connective-tissue sheaths (*S*) and embedded in subendocardial connective tissue (*C.T.*).

network of Purkinje's fibers are enclosed in a continuous connective-tissue sheath. Unfortunately, the system cannot be injected in the human heart, but can be really demonstrated by this method in the beef or sheep heart. The spaces between the Purkinje's fibers and their sheaths, as shown in Fig. 187, do not exist during life. These spaces become evident only on the passage of tissue through dehydrating solutions which shrink the Purkinje's fibers more than the surrounding connective-tissue

sheath. Attempts to inject beef hearts immediately after removal are unsuccessful. Best results are obtained in hearts kept either in the ice-box for twenty-four hours or at room temperature for several hours. It is impossible to state exactly the time interval. It is advisable to take several hearts and attempt the injections at different time intervals. On close inspection the left branch bundle can readily be detected, even

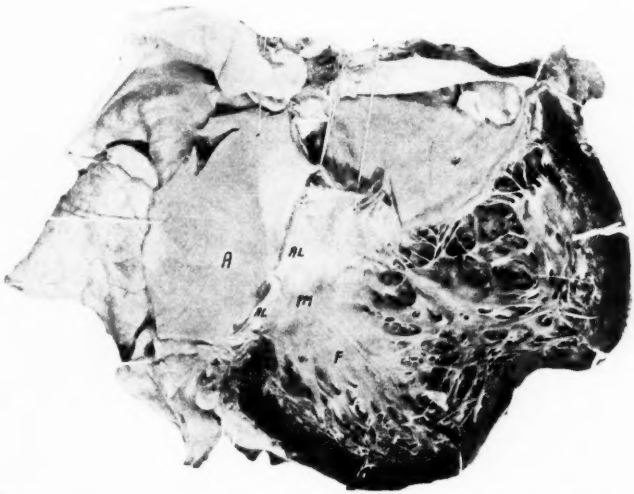

Fig. 188.—Heart showing marked overgrowth of subendocardial connective tissue which partly replaces the specialized cells of the left branch bundle. *A* = aorta. *AL* = aortic leaflets. *P.M.* = pars membranacea of interventricular septum. *F* = area of subendocardial fibrosis.

the finer branches of the network can be seen as delicate slightly yellowish lines through the endocardium. The injections should be started by inserting a fine needle in the left branch bundle. Sometimes the entire system on the left side can be injected through one puncture. If this proves unsuccessful, smaller branches can be picked up and the injection completed. The right bundle can be readily detected after cutting cordæ tendinæ of the septal segment of tricuspid valve and reflecting

it. For unknown reasons it is much more difficult to inject Purkinje's system in the right ventricle.

Briefly, the normal pathway of the cardiac impulse is as follows: The impulse originates in the sino-auricular node, the pace-maker of the heart-beat. From there it spreads over the musculature of the ventricles to the auriculoventricular node. From there it is transmitted through the fibers of the auriculoventricular bundle, its branches, and the network of Purkinje's fibers to the musculature of the ventricles.

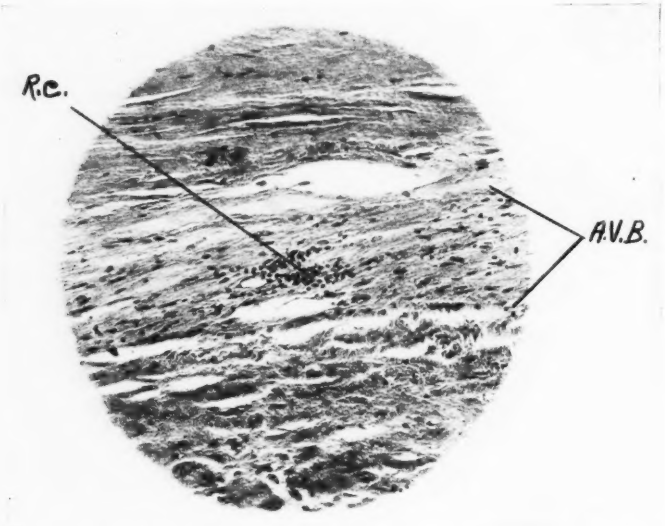

Fig. 189.—Crosss-ection of auriculoventricular bundle from a case of heart-block showing round-cell infiltration of the bundle. *A.V.B.* = auriculoventricular bundle. *R.C.* = round-cell infiltration.

Pathologic changes in the auriculoventricular node, in the auriculoventricular bundle, its main branches or finer arborizations interfere with the conduction of stimuli over their normal pathway and lead to certain types of arhythmias.

Armstrong and Monckenberg, quoted by Lewis, have reported a case of complete heart-block due to an endothelioma destroying the auriculoventricular node.

Lesions of the auriculoventricular bundle lead to either incomplete or complete heart-block, depending on the degree of destruction of this specialized tissue. Complete section always produces complete block. Inflammations and fibrosis of surrounding structures often extend into the bundle and produce complete transverse destruction of the bundle (Figs. 189, 190). Calcareous infiltration, usually extending from calcareous aortic valves, produces similar changes. Less commonly, ulcerations, infection from aortic valves destroy the bundle or its left

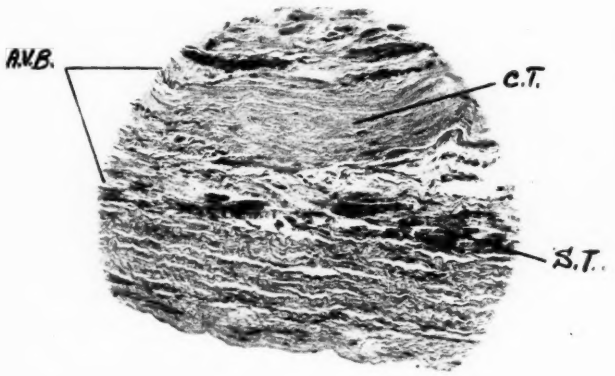

Fig. 190.—Cross-section of auriculoventricular bundle (*A.V.B.*) from a case of heart-block showing extensive replacement of specialized tissue by dense connective tissue (*C.T.*). Only a few fibers of the specialized tissue (*S.T.*) are seen in the section.

branch. Gumma in the region of the bundle is a rather common cause of heart-block. Tumors, mainly endothelioma, are rare. Severe intoxications, such as diphtheria, may lead to toxic changes of the specialized tissue and interfere with normal conduction.

Lesions destroying the left or right branch bundles lead to branch bundle block. Subendocardial fibrosis is a common cause (Fig. 188). Acute mural endocarditis, gumma, and tumors also may destroy the branch bundles.

Subendocardial fibrosis and coronary thrombosis may destroy arborizations over a considerable area and produce arborization block.

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HOW TO TELL THE CARDIAC ARHYTHMIAS AT THE BEDSIDE

JAMES E. TALLEY

WE will take them somewhat in the order of importance and frequency.

Auricular Fibrillation.—The importance of auricular fibrillation is shown by the fact that 60 to 70 per cent. of patients with cardiac failure show this form of total irregularity. In this condition the auricular walls stand in diastole. No systole is accomplished, but the surface of the auricular wall is the seat of rapid undulations due to a sinuous circus movement circulating at an average rate of 450 to the minute. Therefore, instead of one regular impulse from the sino-auricular node stimulating the ventricles at regular intervals, there are a swarm of irregular stimuli. Fortunately, the junctional tissues of the human heart will transmit at most but 200 of these to the ventricle. Ordinarily, the totally irregular ventricular rate clinically varies from 90 to 160, but it may be as low as 40 as under digitalis. Patients with auricular fibrillation may be divided into the rheumatic and the sclerotic groups. We look for the former during the third and fourth decades; the latter from fifty onward. In the sclerotic group it occurs more often in men; because of its common association with mitral stenosis women suffer as much as men in the rheumatic group. A history of persistent irregularity of the heart, coupled with a history of rheumatic fever, chorea, mitral stenosis, chronically enlarged heart, arterial disease, interstitial nephritis, and advanced exophthalmic goiter suggests auricular fibrillation as a cause.

The diagnosis of auricular fibrillation is made at the bedside by a study of the pulse, the apex-beat, and the influence of heart rate on the irregularity. In all the arhythmias, but in this one

in particular the radial pulse is an indifferent sign of ventricular activity, because many beats fail to reach the wrist so that commonly there is a pulse deficit. The irregularity is most evident when the ventricular rate is from 120 to 160. The radial pulse then is a medley of beats of varying strengths and pauses of different lengths. There is a continuous intermingling of normal, weak, strong, lost, and returning beats. There is no relation between the size of the beat and the length of the preceding pause. When the rate is slow, 90 or below, the disorder is less pronounced, is harder to recognize, and instrumental means may be necessary, but a strip of tracing of the radial curve is usually quite sufficient. At the apex the persistent irregularity is even more marked; the sounds vary in intensity with the strength of the beats; first and second sounds are heard in each systole which forces blood into the aorta. If the beat is ineffectual the radial pulse is missed and the first sound at the apex stands alone.

If the veins of the neck are prominent, the jugular pulsations will be found synchronous with the apex-beat. No presystolic wave will be seen because the venous pulse is of the ventricular form. In mitral stenosis with the onset of fibrillation the murmur takes on new time relations. It is practically never confined to presystole and shows no definite presystolic accentuation. Since auricular contraction causes or accentuates the diastolic murmur of mitral stenosis, and since in auricular fibrillation there is no auricular systole, there can be no presystolic murmur. If the murmur of mitral stenosis was rough and well-developed when fibrillation occurred, the murmur becomes early or middiastolic. It usually begins a little after the second sound and runs through to the succeeding first sound or may fall short of it. The second sound at the apex is due to a closure of the aortic and pulmonary valves and they close when the intraventricular pressure falls to the level of the intra-aortic pressure. But no blood can flow from the left auricle into the left ventricle until the intraventricular pressure has fallen below the auricular level. General stasis may lead to incomplete ventricular emptying, and a greater degree of ventricular relaxation may be necessary than normal before this relationship of the two pressures is established. For

this reason the early or middiastolic murmur of mitral stenosis has a distinct relationship to the second sound, but the murmur usually begins at a distinct interval after the second sound. It is this late diastolic murmur at the apex in mitral stenosis with auricular fibrillation which is sometimes confused with the aortic diastolic murmur. Knowing that it exists, the uncommon condition of aortic incompetence with auricular fibrillation should never be diagnosed in the absence of a diastolic murmur at the aortic cartilage and a water hammer pulse. If one studies the published phonocardiograms of heart sounds and murmurs, especially in mitral stenosis both with regular rhythm and fibrillation, this whole matter is more easily understood.

Studying the relation of this irregularity to heart rate is a simple and certain means by which auricular fibrillation may be recognized clinically. Moderate exercise, amyl nitrite, or the presence of fever will increase the heart rate whether we have a regular sinus rhythm or fibrillation. With the increase of heart rate, especially to 120 or above, the irregularity always increases in fibrillation; whereas the irregularity due to extra systoles, partial heart-block, or sinus arhythmia is wiped out by the increase in heart rate. Again, as the heart rate slows—as at rest after exercise—the ventricular irregularity in fibrillation grows less; whereas with rest, irregularity due to extra systoles or partial heart-block returns. These tests apply especially to patients with slow fibrillation, with but few or no signs of heart failure. A persistent ventricular irregularity in a patient with evident signs and symptoms of serious heart failure, especially if the heart rate is high, is almost always due to auricular fibrillation. Indeed, when a ventricle beats irregularly at 120 or above, the irregularity is almost always due to auricular fibrillation. Again, auricular fibrillation once established is usually persistent year after year, though, of course, we are all acquainted with its paroxysmal form.

Premature Beats or Extrasystoles.—They may be ventricular, auricular, or nodal. They can be differentiated absolutely only by instrumental means, but, fortunately, this is not important clinically. They are extremely common. It has been said that

everyone who lives to middle life or beyond has probably had them at some time. They occur in well-recognized cardiovascular disease as well as in the apparently normal heart. They may be precipitated or aggravated, especially in those predisposed by tobacco, tea, and coffee. When the individual is fatigued physically and nervously, these three agents more easily precipitate this irregularity. They are rare where the heart rate is 100 and practically unknown where the rate exceeds 120. Exercise will increase the heart rate and abolish this form of irregularity, but as the heart slows following the exercise they are apt to return. They may be present in an individual while standing and disappear when he is recumbent.

When a single premature beat interrupts the regular rhythm, an early cardiac pulsation will be recognized at the apex with two extra sounds, if the aortic valves are opened, also a weak premature arterial pulse. If the premature beat is too weak to affect the arterial circulation, there willl be an intermission at the wrist and one extra sound at the apex. The bigeminal, trigeminal, and quadrigeminal pulse is usually due to a premature beat taking place of each second, third, or fourth beat of a rhythmic series. This premature beat is usually ventricular, occasionally it is auricular.

If one has developed an acute time sense, it may be possible to say that the premature beat is of ventricular or auricular origin. With a ventricular premature beat the whole period of disturbance is equivalent to two beats of the normal rhythm. If the premature beat is auricular, the period is usually short of this. When a premature ventricular beat coincides with an auricular contraction of the rhythmic series, the two heart chambers are in systole together and a prominent swelling of the veins of the neck will be the result, because for a single cycle the auricle cannot empty into the ventricle and there is a reflux wave. For the same reason the first sound may be occasionally accentuated. One point in treatment I cannot refrain from emphasizing. In individuals especially disposed, when tired physically and nervously, at times the persistence of premature beats on retiring has been distressing and sleep destroying. In these in-

dividuals quinidin sulphate in dosage of 2 to 3 grains, three times daily, and sometimes less often has finally abolished the premature beat and brought comfort. Some of the patients have had to continue the remedy for months; after prolonged vacation with mental rest and needed physical exercise they have been free from premature beats of which they were conscious.

Pulsus Alternans.—In alternation of the ventricle the heart rhythm is regular, but the beats alternate in strength, a strong and a weak beat following each other regularly. Thus, the left ventricle expels larger and smaller quantities of blood at alternate contractions. It is a common condition where the apex rate is extremely rapid as in paroxysmal tachycardia. It frequently is revealed only for a few beats after an extrasystole, but it is the constant alternation with normal heart rate that is especially important. It is found associated with angina pectoris, with high blood-pressure, with nephritis, myocardial degeneration, and sometimes in pneumonia and after digitalis. Its detection by palpation of the pulse or apex and auscultation of the latter is rarely possible. The best means of diagnosis at the bedside is by using the blood-pressure apparatus. There is a difference of a few millimeters of mercury between the pressures at which the large and small beats come through. By carefully adjusting the cuff and by slowly raising and lowering the column of mercury, one can halve or double the pulse rate at the wrist. Also, the difference can be recognized in the sounds in the vessel with the stethoscope at the elbow. The polygraph curve of the radial or brachial depicts this variation in intravascular pressure best of all.

Heart-block.—The term "bradycardia" should be limited to a slowing of the whole heart. The apex-beat, the movement of the vessels in the neck, and the radial pulse maintain their normal relationship. In heart-block the ventricular response to the auricular impulse is delayed or it fails entirely. The first stage of block is that of prolonged conduction. It is rarely satisfactorily demonstrated except by instrumental means. However, where a patient is known to have had rheumatic fever, chorea, syphilis, or to be under the influence of digitalis, a reduplication

of the heart sounds is suggestive of the existence of delayed conduction. The contraction of the auricle produces a muffled sound. The first sound of the heart is made up of this auricular sound plus the more evident result of ventricular contraction. With prolonged conduction of moderate degree the sound of auricular contraction may be sufficiently separate to lead to reduplication of the first sound. If the delay in conduction is more marked, the auricular sound will fall in early diastole and lead to a reduplication of the second sound. The next stage in heart-block is the dropping of a single beat for a single cycle, then there will be silence at the apex and no pulse at the wrist. Every third or fourth beat may be dropped, then the pulse will be grouped in twos and threes and there will be a ventricular silence after each third or fourth beat. Exercise, or if the patient cannot be moved, a few whiffs of amyl nitrite will wipe out the irregularity, but with the fall of pulse rate the irregularity is apt to return. A weak premature beat that does not open the aortic valve also gives origin to a lack of radial pulse beat. In the case of the premature beat a weak movement will be heard at the apex, while in case of block there will be no ventricular contraction.

Two to one heart-block is the stage where there is but one ventricular response to each two auricular contractions. Usually there is a regular ventricular rate of about 40. A sudden exact halving of the ventricular rate which abruptly doubles under exercise is suggestive. In complete heart-block the auricles and ventricles beat entirely independently. The ventricle usually beats perfectly regularly at a rate from 28 to 35. The auricles at fully double this rate. The vagus has little influence over the ventricular rate in complete heart-block so that ordinarily exercise, amyl nitrite, or atropin do not have the usual influence in increasing the heart rate. Each ventricular contraction is usually accompanied by first and second sounds. Sometimes in thin-chested individuals especially the muffled auricular sound can be heard in the long intervals between the ventricular contractions. A continuous change in the intensity of the first and second sounds from beat to beat is suggestive of this condition. The

relatively rapid auricular contractions may be evident in the jugular pulse. Periodic variations in the venous pulsation is as suggestive as the change in the intensity of sound spoken of above. They both depend upon whether the auricular contraction falls just before, with, just after, or stands isolated from, the ventricular contraction. Ordinarily speaking partial block is an unstable condition, whereas complete heart-block when once established is apt to be permanent. There are two phases of heart-block which lead to the Adams-Stokes syndrome. One of the dangerous times is when partial heart-block is passing to complete heart-block and then back to the lower grade again. Occasionally a patient will have for some weeks rates of 80, 40, and 30, changing from day to day, accompanied by fainting, unconsciousness, and mild fits. These rates mean that he has passed in quick succession from regular rhythm to two to one heart-block, and then to complete block, and then again reversing the order. Occasionally, one sees such a patient settle permanently into complete heart-block with a ventricular rate of 30 and continue thus for years, since he is fortunate in having sound ventricular muscles. Again, in complete heart-block with a ventricular rate of 30, in certain patients there is a sudden slowing of the rate to 20, 12, or 8, which leads to the full-blown syndrome.

Simple Paroxysmal Tachycardia.—The term of "simple tachycardia" should be limited to hearts beating rapidly in response to impulses arising in the sino-auricular node. It is the normal physiologic acceleration found after exercise and in fevers. Simple paroxysmal tachycardia, on the other hand, is an abnormal rhythm. It is in response to an impulse arising in an ectopic and pathologic focus. It is like a continuous series of premature beats; it may arise in the auricle or the ventricle. It may last for a few seconds, minutes, hours, or two weeks. The extreme limits of heart rate are 110 to 220; the usual rate is from 160 to 200. Counts at the apex alone are reliable. The onset of the rapid rate and its offset are abrupt. There is none of the gradual increase and gradual decrease of rate such as is found in simple tachycardia. Ordinarily, one will find an apex rate of 180 or

more that is perfectly regular and entirely undisturbed by change of posture. The constancy of the count at various examinations is striking. The variation is never more than five beats from count to count. Effort, holding the breath, change of posture have no influence on the rate. Vagal pressure in the neck or pressure upon the eyeball will do one of two things: it will have no effect at all or it will bring about an abrupt slowing. There will be no gradual slowing, such as there is in simple tachycardia. Anginal pain and hyperalgesia of the left chest muscles are extremely common in this disease. In no other condition can one follow more accurately the dilatation of the heart than comes under the prolonged fast rate nor its rapid recovery after the sudden offset. Attacks running into hours and beyond always lead to alimentary symptoms and congested liver and lungs. It is to be hoped that the intravenous use of quinidin sulphate may prove a valuable remedy where others have failed to stop the attack. Levine and Blatner have recently suggested full digitalization of the patient to prevent the recurrence of attacks.

Auricular Flutter.—In this condition the auricles beat at a rate of 240 to 320—more often from a rate of 260 to 320. This rapid rate is due to a circus movement which revolves around the vena cava. It is a smooth regular wave which keeps to one plane, so that the ventricle receives rapid regular impulses. But since a ventricle could not beat long at the rates given we usually find it protected by some degree of heart-block. Ordinarily, when the patient is first seen, there is a condition of two to one heart-block, so that the ventricular rate is from 130 to 160. Two to one, four to one, and complete block are possible. The suggestive things about its diagnosis are the long duration and constant ventricular rate of 130 to 160. It lasts for months and years unchanged. It occurs more often in elderly men. This rapid rate is unchanged by posture, rest, or exercise. Vagal pressure may slow the ventricle, but has no influence on the auricle. The rapid undulations of the veins of the neck will continue uninfluenced by vagal pressure. It is distinguished from simple paroxysmal tachycardia because in the latter the rate is higher—160, 180, 200—it has not the abrupt onset and offset of simple paroxysmal tachy-

cardia. With higher grades of block flutter may exist, but can be demonstrated only instrumentally. Occasionally, one will find a perfectly regular apex-beat of 75 to the minute, whereas the curve will show that the auricle is running 300 to the minute. Complete block with auricular flutter also occurs. A regular ventricular rate of 30 with a rapid undulation in the veins of the neck would be suggestive.

Sinus irregularities need interest us only in differentiating them from the irregularities already discussed. The sinus arhythmias are largely confined to childhood, but they do occur in nervous individuals in the second and sometimes the third decade. The whole heart is involved, so that the usual relations between apex-beat, vessels in the neck, and radial pulse are maintained. They are usually connected with respiration. Inspiration is usually accompanied by an increase in pulse rate; expiration with a slowing. In an occasional patient there is a long pause in the pulse after each expiration. It simulates a dropped beat, but a dropped beat is not constantly associated with the expiratory phase of respiration.

THE VALUE OF THE POLYGRAPH IN CARDIAC DIAGNOSIS[1]

THOMAS M. MCMILLAN

THE polygraph need hardly be explained, so well known is this compact little instrument. In brief, the ordinary polygraph consists of two air chambers covered with light rubber tissue and connected by rubber tubing to suitable receivers that are applied to the jugular bulb in the neck and radial artery at the wrist. The pulse waves or changes in pressure in these vessels impart movement to the air-column in the tubing and chamber, and move the rubber coverings of the latter. This slight movement is then made to act upon very light and finely balanced pens, and thus the pulse waves of the jugular and radial are recorded in ink upon a moving strip of paper. Such is the ordinary ink polygraph.

Delicately as these little instruments are made, obviously there must be a considerable lag and inertia in such a system.

The same principle is made use of in an exceedingly more sensitive and accurate instrument that employs Frank capsules instead of the ordinary polygraph tambours. These capsules differ from the usual tambours in being considerably smaller, and instead of having the small movements of the rubber covering push relatively heavy pens, a small mirror is put on the side of the membrane and strong light is then focused on the mirrors. The beam of light thus caused is then directed into the lens of a camera, such as is used for electrocardiography and the movements of the beam of light recorded upon moving sensitized paper or film.

Such a system is obviously much more sensitive than an ink polygraph and will record the movements of the veins and arteries

[1] From the Department of Cardiology of the Philadelphia General Hospital.

much more clearly. But such an instrument can be used only in a hospital or laboratory. This fact vitiates the chief clinical advantage of the polygraph—its easy portability.

The Waves of the Polygram.—In correctly taken polygrams there are several landmarks that must be clearly seen if the tracing is to be adequate for interpreting correctly the various events of the cardiac cycle and for determining the mechanism of the heart.

In Fig. 191 (taken from Lamson, The Heart Rhythms, Williams & Wilkins Co., Baltimore, 1921, p. 74) are diagrammatically represented the waves of the normal polygram and the corresponding events in the heart. From this it can be seen that there are two landmarks in the radial tracing that are necessary for the identification of the waves of the jugular pulse. The beginning of ventricular systole as shown in the radial tracing is preceded by one-tenth second by the conspicuous "C" wave of the jugular. The dicrotic notch of the radial occurs synchronously with the "V" wave of the jugular and must be clearly seen for the correct identification of the latter. With these two jugular waves identified, the recognition of the only other jugular wave in the normally beating heart, the "A" wave, is relatively easily accomplished by picking out the wave that precedes the "C" wave by approximately one-fifth second.

The origin and causation of these various waves have been searchingly investigated by various workers; even now some of the finer physiologic facts are still in doubt.

The upstroke of the radial is caused by the onrushing wave of blood that is set up by the injection of the left ventricular contents into the aorta. The mechanism of the formation of the dicrotic notch is still not entirely understood. Suffice it is to say that it occurs synchronously with closure of the aortic valves, and therefore in the polygram marks for us the end of ventricular systole. It may be looked upon from a clinical viewpoint as due to a "back kick" (Lamson) of blood that follows upon the fall of aortic pressure incident to ventricular diastolic relaxation.

The "A" wave of the jugular accompanies auricular contrac-

tion. The jugular "C" was given its name because it was thought to be the result of pulsation of the underlying carotid artery. Closer investigation has thrown some doubt upon the accuracy

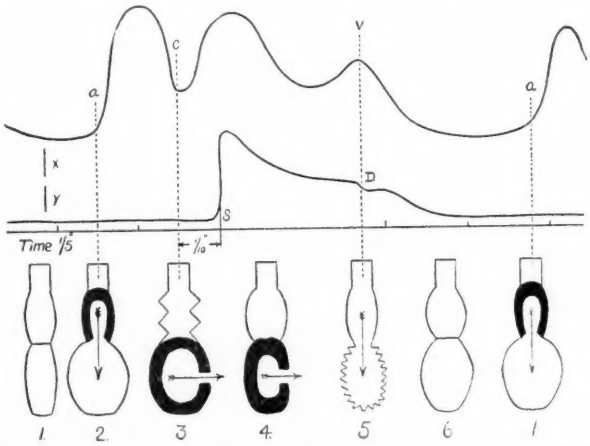

Fig. 191.—Diagrammatic scheme showing the cause of the venous and arterial pulse waves. At (*1*) both auricle and ventricle are partially filled with blood. At (*2*) the auricle contracts as shown by its heavy outline and forces blood into the ventricle, distending it. At the same time it sends a wave up the veins to the neck and causes a rise in the venous curve, the *a* wave. At (*3*) the auricle relaxes as shown by the irregular outline and the ventricle contracts and drives the blood out into the aorta. This contraction causes a wave in the venous curve, the *c* wave at the beginning of systole. The ventricular pulse wave does not reach the wrist until one-tenth second after the corresponding wave in the neck, due to the time taken to travel there. At (*4*) the ventricle is still contracting and the auricle filling with blood. This causes backing up of blood in the veins and a rise in the venous curve. At (*5*) the ventricle relaxes, end of systole, and the blood falls from the auricle into the ventricle causing a sharp drop in the venous curve, the *v* wave (really a drop in the curve). At the same time the dicrotic notch occurs in the arterial pulse. At (*6*) both the auricle and ventricle are passively filling with blood, and at (*1*) the cycle is begun again by contraction of the auricle. (After Lamson, The Heart Rhythms, Williams & Wilkins Co., Publishers.)

of this interpretation. At any event it undoubtedly is the result of ventricular systole—perhaps this contraction transmitted through the floor of the auricle, the chamber itself, and thence up the vein. This wave precedes the radial upstroke simply

because it is nearer the origin of the wave. During ventricular systole the pressure within the auricle rises. As the ventricle relaxes this increasing auricular pressure falls with the discharge of blood from the auricle into the ventricle. These changes result in the production of the "V" wave of the phlebogram.

In Fig. 192 are shown four different ink polygrams. The bottom tracing is a mirror polygram taken with Frank capsules. (The time on all ink polygrams is in one-fifth second; on all mirror tracings, it is in one-fifth and one-twenty-fifth second.)

THE INTERPRETATION OF ABNORMAL CARDIAC RHYTHMS BY THE POLYGRAPH

Sinus Arhythmia.—This physiologic arhythmia is recognized by the slight irregularity in the occurrence of "A" waves. Each "A" wave is followed by normally sequential "C" and "V" waves.

Extrasystoles.—In auricular extrasystoles it can be seen in the radial that there are occasional premature beats. In the jugular it can be seen that the premature event is the "A" wave. The "C" and "V" waves follow the "A" waves normally (Fig. 193). In ventricular extrasystoles again the radial reveals the presence of premature beats. On studying the beats in the jugular it is seen that the premature events are "C" waves; the regularity of the "A" waves is undisturbed. If the premature "C" wave occurs synchronously with the normal regular "A" wave, as it usually does, we have these two waves shown in the polygram by one large summated wave. The important thing to discover is whether the hidden "A" wave is occurring in its normal sequence (Fig. 193). In Fig. 193 are shown two examples of ventricular and one of auricular extrasystoles. The last two strips of this figure are electrocardiograms of ventricular and auricular extrasystoles. These are put in to show how much more certainly the electrocardiograph shows these disturbances. In nodal extrasystoles we have the auricular and ventricular waves, the "A" and "C" waves occurring synchronously. This results again in a large summated wave and resembles a ventricular extrasystole. The difference lies in the fact that the "A" wave

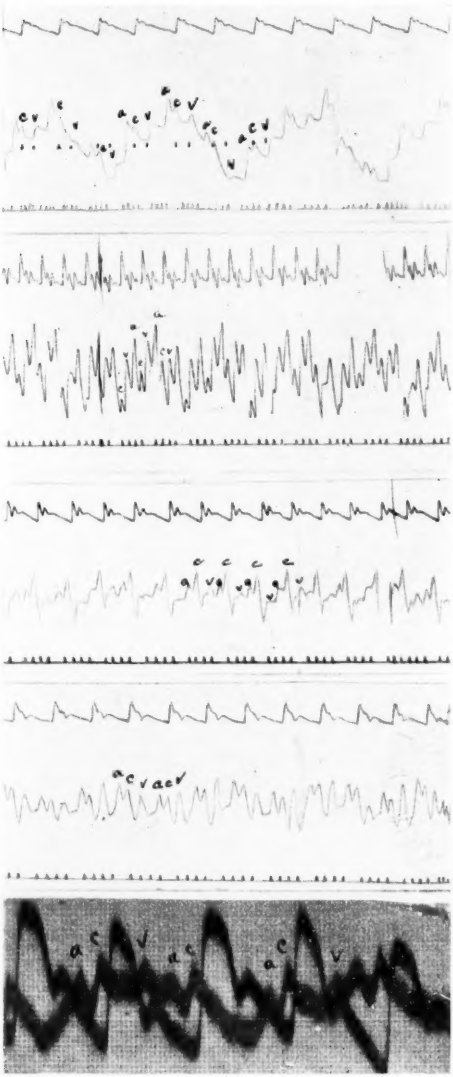

Fig. 192.—The first four strips are normal ink polygrams. The bottom strip is a normal radial and jugular mirror tracing obtained with Frank capsules.

31 81

is usually slightly out of its rhythm with the normal "A" waves (Fig. 194).

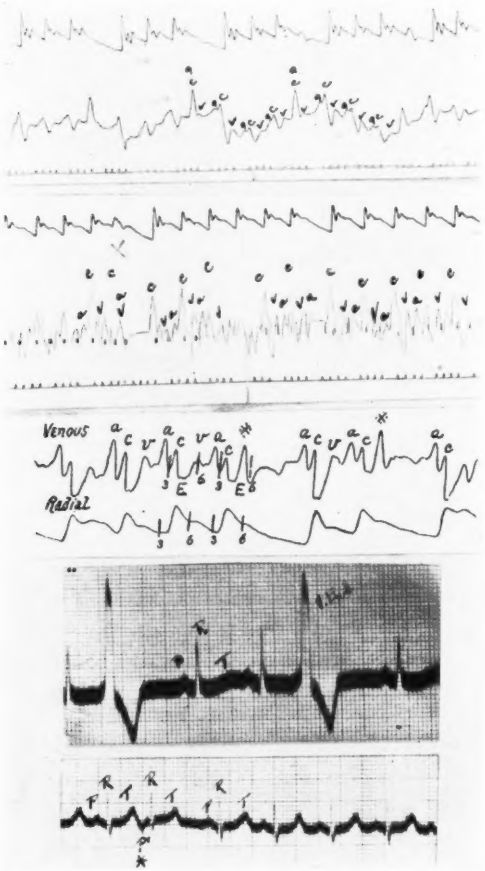

Fig. 193.—The first two tracings illustrate ventricular extrasystoles as shown in the polygram. The third tracing, taken from Lewis, are electrocardiograms of ventricular and auricular extrasystoles. These are included to show how much more clearly and certainly these disturbed impulses are shown by the electrocardiograph. (Strip 3 from Lewis, The Mechanism and Graphic Registrations of the Heart-beat, Shaw & Sons, Ltd., London, Publishers.)

Nodal Rhythms.—In this disturbed rhythm all "A" waves are occurring synchronously with "C" waves and are therefore hidden. It is true the composite summated wave will be a large

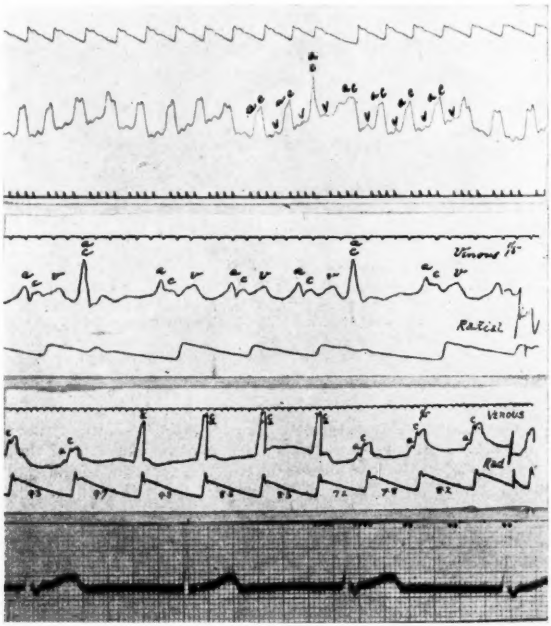

Fig. 194.—The second and third polygrams show respectively nodal extrasystoles and nodal rhythm. Both these are taken from Lewis. The first tracing shows a ventricular extrasystole. In this tracing and in the third tracing are high waves caused by the summation of *a* and *c* waves. In the tracing of nodal extrasystoles it can be seen that both the *a* and *c* waves are premature. In the tracing of ventricular extrasystoles the *c* wave alone is premature. The last tracing is an electrocardiogram of nodal rhythm. (Strips 2 and 3 from Lewis, The Mechanism and Graphic Registration of the Heart-beat, Shaw & Sons, Ltd., London, Publishers.)

one, but it is difficult to say that this results from summation and is not the result simply of a sensitive membrane. The generally slow rate and the absence of independent "A" waves is confirmatory evidence of the diagnosis (Fig. 194).

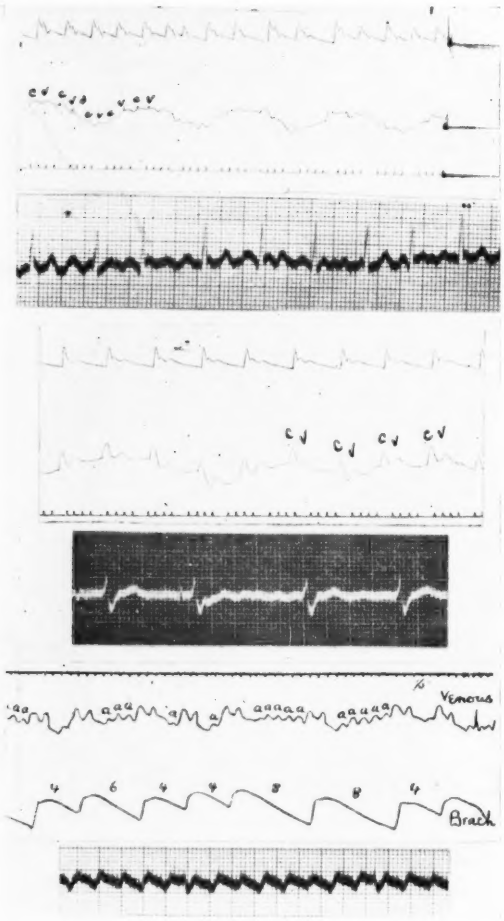

Fig. 195.—The two upper polygrams are from cases of auricular fibrillation. Each is accompanied by an electrocardiogram of the same case. The bottom polygram, taken from Lewis, is from a case of auricular flutter. Below it is an electrocardiogram of auricular flutter (not the same case). The diagnosis is made in all instances, more certainly by the electrocardiogram than the polygram. (Strip 5 from Lewis, The Mechanism and Graphic Registration of the Heart-beat, Shaw & Sons, Ltd., London, Publishers.)

Auricular Fibrillation.—In the polygraph we find no "A" waves. This and the irregular ventricular action as seen in the radial usually establish the diagnosis (Fig. 195).

Auricular flutter reveals itself by showing rapid regular "A" waves occurring more rapidly than the ventricular waves and bearing some mathematic proportion to them (Fig. 195).

The **tachycardias** are usually difficult of analysis in the polygram.

Auricular paroxysmal tachycardia gives the same picture as simple tachycardia, unless one is so fortunate as to secure the beginning or ending of a paroxysm.

Ventricular paroxysmal tachycardia shows rapid regular "C" and "V" waves without preceding "A" waves. Slower auricular waves occurring independently of ventricular waves may be seen. As a matter of fact the differentiation of the tachycardias by the polygraph is quite uncertain usually.

A-V heart-block can usually be easily analyzed from polygrams. The first stage is recognized by a prolonged a-c interval. The second stage of dropped ventricular beats is shown by "A" waves not followed by "C" and "V" waves. Complete block is recognized by the regular ventricular beats, occurring more slowly and independently of the more rapid auricular beats (Fig. 196).

The Difficulties of the Polygraph.—The polygraph is a difficult record both to obtain and to interpret. A considerable degree of skill, or perhaps better, knack and practice is necessary to obtain satisfactory tracings. The necessity of more or less constant practice is illustrated when one attempts a tracing after having laid aside the instrument for a time. The interpretation of polygrams is a very difficult matter and requires considerable study and experience. It is a much more difficult matter than interpreting electrocardiograms and I believe an acquirement that is obtained with more difficulty.

Comparison of the Relative Value of Polygrams and Electrocardiograms.—It is only frank to say that the polygraph as an instrument for analyzing cardiac arhythmias is losing its vogue. This is because of the development of the electrocardiograph. This latter method is simpler and is infinitely more certain.

After interpreting a polygram there very often remains considerable doubt of the correctness of one's conclusions. The interpretation of the electrocardiogram is usually definite and certain.

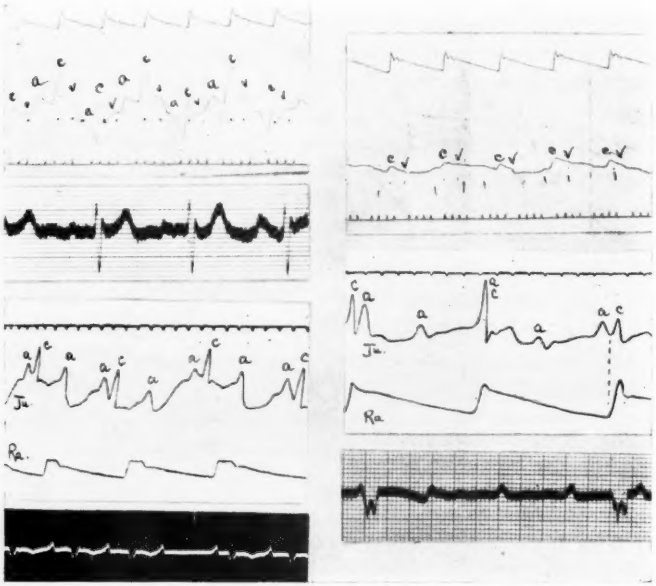

Fig. 196.—The first polygram on the left and the accompanying electrocardiogram are both from a case of heart-block with prolonged *a–v* conduction time. The polygram under this, taken from Lewis, shows a high grade of heart-block in which every second auricular beat fails to be followed by a ventricular response. The accompanying electrocardiogram shows a single dropped ventricular beat. The two polygrams on the right, the latter after Lewis, are from cases of complete *a–v* block. The accompanying electrocardiogram is also from a case of complete heart-block. (Strips 3 and 6 from Lewis, The Mechanism and Graphic Registration of the Heart-beat, Shaw & Sons, Ltd., London, Publishers.)

The electrocardiogram, with two or more notable exceptions, gives all the knowledge that a polygram gives, and gives us a mass of information that the polygraph fails to reveal. The development of a portable electrocardiograph, a not distant accomplishment, will almost entirely displace the polygraph as a graphic

means of studying the cardiac irregularities. However, the polygraph will always have a place in studying the circulation, for it

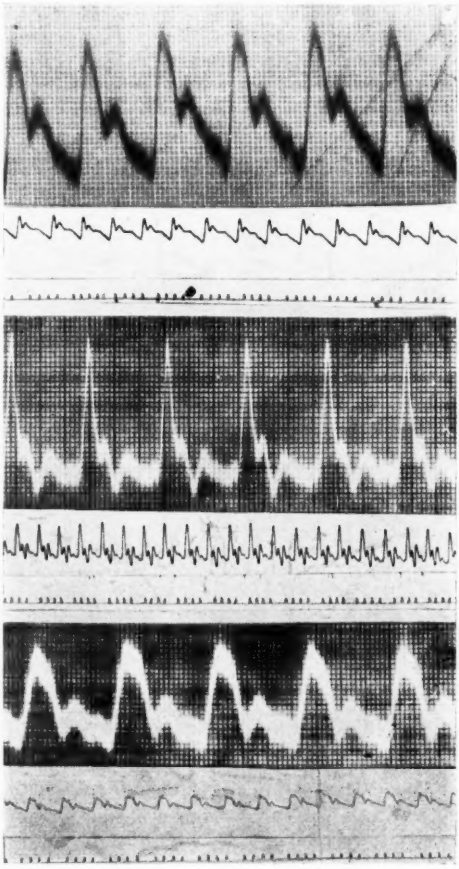

Fig. 197.—Radial pulse waves. The three mirror tracings are accompanied by ink tracings. Normal, collapsing, and sustained or plateau pulses are shown. These features are very much more clearly shown by the mirror tracings.

reveals some things that the electrocardiograph or any other means of graphic examination does not show.

The findings that the polygraph is indispensable in showing: There are a few conditions which will be revealed by the polygraph only or rather by radial pulse tracings. One of these is pulsus alternans. Another is pulsus paradoxus, a condition that is said to have some value in establishing the diagnosis of pericardial pathology. Still another and very valuable use that the study of the arterial pulse wave can be put to is in determining the collapsing or sustained quality of the radial pulse: Determinations of importance in determining the presence or absence of aortic insufficiency and stenosis.

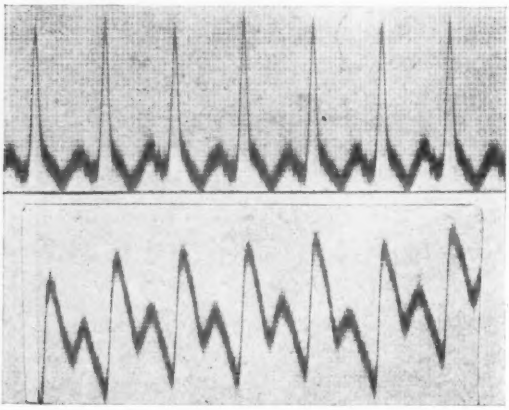

Fig. 198.—(See text.)

In Fig. 197 are shown mirror and ink polygrams of normal, collapsing, and sustained radial pulses.

In Fig. 198 are shown mirror radial tracings from a single case of beriberi. In the first strip, taken at the height of the disease, a typically collapsing pulse is shown. The blood-pressure and other phenomena were characteristic. The second strip was obtained after two months' treatment. This shows a normal radial pulse tracing. At this time the blood-pressure had returned to normal and the water hammer quality of the radial pulse had disappeared. These changes are shown nicely in the pulse tracings.

In Fig. 199 are shown mirror radial tracings that illustrate pulsus alternans and pulsus paradoxus.

Its value in graphically recording these and other conditions, as well as its value in pure physiologic investigation of circula-

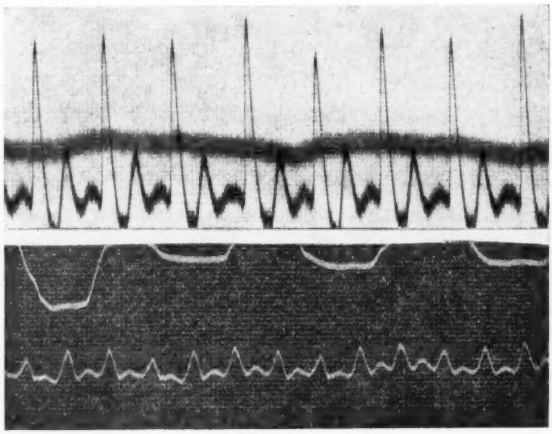

Fig. 199.—The top tracing shows alternation of the radial pulse. The lower tracing from a case of massive pericardial effusion shows "pulsus paradoxus." The respiration curve is shown at the top. It moves up during inspiration. The pulse waves during this phase are smaller. This characteristic disappeared following the removal of 800 c.c. of fluid from the pericardial sac.

tory phenomena, will always assure the instrument of an important place in the study of the circulation, though it probably will be largely superseded by the electrocardiograph, when the instrument becomes portable, in analyzing the arhythmias.

ACUTE ENDOCARDITIS

GEORGE W. NORRIS

IN considering the condition—acute endocarditis—it is perhaps well to emphasize the fact that we shall endeavor to abide by our assignment, ignoring as much as may be such frequently accompanying lesions as myocarditis and pericarditis as well as their fibrotic sequelæ. Too frequently endocarditis is confused with valvular disease which follows it; and too frequently the chronic valvular lesion is blamed for the decompensation which is the result of an acute inflammatory process.

Endocarditis may not involve the valves and may not cause murmurs if it does, whereas murmurs may arise from many other causes.

Classification is unsatisfactory. It is customary to speak of simple or benign as contrasted with malignant types. The term benign is highly objectionable. There is no "benign" endocarditis. Furthermore, different types often merge. Classification based upon pathologic anatomy such as verrucous, ulcerative, perforating is for clinical purposes useless. Obviously our nomenclature should be based upon the infecting organism, but only too often we are ignorant in this respect to say nothing of the fact that the causative factor of rheumatic fever still remains undiscovered.

In regard to *pathogenesis* three views obtain: (1) The organisms lodge on the valves and are mechanically forced into the endocardium. (2) The organisms reach the valves through their own blood-vessels, which we now know exist at least in the mitral and tricuspid valves.[1] (3) Infection occurs via the lymphatics.

[1] Kerr, W. J., and Mettier, S. R.: The Circulation of the Heart Valves, Amer. Heart Jour., 1925, i, 96; Kugel, M. A., and Gross, L.: Ibid., i, 1926, 304.

Swift[1] has suggested that just as spirochætes may lie dormant in the cornea—later become active—cause inflammation and vascularization, both of which conditions may again disappear, only, however, to more readily reappear under favorable circumstances; so endocarditis may arise from lymphatic infection. This condition of vascularity, a sequel of rheumatic endocarditis, explains the frequent infection of old scarred valves by other micro-organisms.

SIMPLE ENDOCARDITIS

Simple endocarditis, a mild verrucous type due to superficial necrosis and thrombosis of a given—usually valvular—area, may occur in chronic nephritis, in some cachexias, and in some acute infections. It may be suspected, but is rarely diagnosticated with any degree of certainty.

RHEUMATIC ENDOCARDITIS

Rheumatic endocarditis, the commonest as well as the most important variety of endocarditis, is the condition which makes rheumatic fever the dangerous plague that it is. Rheumatic fever in children is nearly always associated with heart infection. Indeed some of our best clinicians today no longer consider endocarditis as a complication of rheumatic fever, but as an integral part of the disease itself.

Etiologically we are still in darkness. A non-hemolytic streptococcus is sometimes cultured, but most students of the subject still feel that the real virus has as yet escaped identification. Clawson[2] believes that the streptococcus belongs to the viridans group and suggests that "rheumatic endocarditis and subacute bacterial endocarditis may be produced by the same agent and are but different degrees of the same process."

Clawson and Bell[3] point to the difficulty of differentiating

[1] Swift, H.: Nelson's Loose Leaf Living Med., iv, 206.

[2] Clawson, B. J.: Jour. Infec. Dis., 1925, xxxvi, 444.

[3] Clawson, B. J., and Bell, E. T.: A Comparison of Acute Rheumatic and Subacute Bacterial Endocarditis, Arch. Int. Med., 37, lxvi, 1926.

between rheumatic endocarditis and mild cases of the subacute bacterial variety. They state "The leukocyte count is of little value in differentiating rheumatic from subacute bacterial endocarditis; that a severe secondary anemia strongly suggests the subacute bacterial type; that the order of frequency of valvular involvement is essentially the same in both forms; that in both types there is a proliferative inflammation in the valve, but the thrombus over the infected area is larger and softer in the subacute lesions; that the differences are in degree rather than in kind; that typical rheumatic vegetations may be found with subacute vegetations on the same leaflets; that myocarditis is more frequent in the acute rheumatic cases that come to necropsy; that pericarditis seems to occur with practically the same frequency in rheumatic and in subacute bacterial endocarditis; that the frequent presence of embolic phenomena in the subacute bacterial type depends on the degree of involvement of the infected valves; that mural endocardial involvement occurs with about the same frequency in both types, but is more extensive in the subacute bacterial; that the finding of bacteria in the bloodstream in endocarditis does not necessarily indicate the presence of the condition clinically recognized as subacute bacterial endocarditis; that experimental endocarditis and myocarditis—similar to rheumatic lesions can be produced in rabbits by injecting streptococci; and finally that the streptococci, generally the viridan strains, seem to be responsible for both rheumatic and subacute bacterial endocarditis." This view is not generally accepted.

Congenital heart lesions are notoriously likely to secondary streptococcic infection. Abbott[1] has collected 555 such cases. Secondary lesions are found especially in septal defects and upon malformed cusps.

The relationship between foci of infection and endocarditis and myocarditis is generally accepted. The question as to whether bacteria localize selectively cannot be so certainly stated, although we know that they sometimes do. This has

[1] Abbott, M. E.: Incidence of Bacterial Inflammatory Process in Cardiovascular Defects: Malformed Semilunar Cusps, Amer. Clin. Med., iv, 89, 1925.

been shown to occur both clinically and experimentally and to be independent of other factors, such as injury, fatigue preceding disease, etc.[1]

Experimentally the severity of endocarditis can be made to vary not only by increasing the virulence of the organism, but also by decreasing the resistance of the host.

That the tonsil is not the only source of infection in rheumatic fever is indicated by Shaw's[2] observations in the British Navy in which there was a marked decrease in rheumatic fever in 1921 while the tonsillitis rate remained stationary. Indeed Grenet[3] believes that the portal of entry may sometimes be the gastro-intestinal tract inasmuch as in a number of his patients rheumatic infection was preceded by diarrhea.

The **pathology** of rheumatic endocarditis, its verrucous character, its tendency to chiefly involve the mitral valve and to be renewed and progressively damaging with each succeeding attack are well known. Microscopically variable degrees of thrombosis, hyalinization, focal necrosis, cellular infiltration, vascularization, and granulation are seen. The most characteristic lesion, however—the Aschoff body—occurs in the accompanying myocarditis. Myocarditis deserves emphasis, both on account of its frequency and because both early and late it is the myocardial rather than the valvular damage which works havoc with the heart.

The importance of myocardial infection and the frequency with which it is directly accountable for a lethal outcome has been aptly emphasized by Thayer.[4] The Aschoff body is a perivascular collection of peculiar large cells containing rounded vesicular nuclei with granular protoplasm with characteristic

[1] Nickel, Allen C.: Localization in Animals of Bacteria Isolated from Foci of Infection, Jour. Amer. Med. Assoc., 1926, lxxxvii, 1117.

[2] Shaw, T. B.: Remarks on the Incidina of Aetiology and Prevention of Rheumatic Fever in the Navy, Proc. Roy. Soc. Med. (War Section), 1925, xviii, 9.

[3] Grenet: Digestive Symptoms in Acute Articular Rheumatism, Progress. Med., 1925, No. 12, 89.

[4] Thayer, W. S.: Acute Rheumatic Disease of the Heart, Bull. Johns Hopkins Hospital, 1925, xxx, 99.

staining qualities. It occurs only in rheumatic fever and chorea, usually in the left ventricle, and has not thus far been experimentally reproducible.

Papenheimer and von Glahn[1] have called attention to rheumatic aortitis characterized by Aschoff bodies in the adventitia and dense scarring of vascular distribution. Histologic pictures closely resembling Aschoff bodies have been found in the valves and in the pericardium.

Aschoff bodies have also been found in association with marked left auricular thickening in the region above the mitral leaflet—a thickening not due to the usual fibrin deposit, but rather to a tissue hyperplasia.[2] They have also been found in the brain in chorea.

Evidences of Rheumatic Endocarditis.—It is always safest to suspect if not actually to assume the presence of cardiac infection in every case of rheumatic fever. In children it is constantly present often with slight, and at times without any, involvement of the joints. A preceding tonsillitis, either acute or chronic, is usually present. In chorea, the third and worst of the rheumatic triad, the heart rarely if ever escapes infection and usually suffers structural damage. Careful examination of other members of the family shows one or more of the three above-mentioned conditions present in about the same proportions as evidences of tuberculosis are found among the relatives of a tuberculous individual (14 per cent.) (St. Lawrence).[3]

In an attempt to study the increased liability of members of "rheumatic" families to rheumatic infections, Faulkner and White[4] investigated the subjects of heredity, environment, and direct contagion. The first topic, heredity predisposition, yielded no conclusive results. The study of environment showed

[1] Papenheimer, A. M., and von Glahn, W. C.: Lesions of the Aorta Associated with Acute Rheumatic Fever, Jour. Med. Research, 1924, xliv, 489.

[2] MacCallum, W. G.: Rheumatic Lesions of the Left Auricle of the Heart, Bull. Johns Hopkins Hosp., 1924, xxxiv, 524.

[3] St. Lawrence: Jour. Amer. Med. Assoc., 1922, December 16th.

[4] Faulkner, J. M., and White, P. D.: The Evidence of Rheumatic Fever, Chorea, and Rheumatic Heart Disease, Jour. Amer. Med. Assoc., 1924, lxxxiii, 426.

much less rheumatic infection among the so-called upper classes. The third topic infection, on the other hand, showed that nearly 9 per cent. of the parents of children recently infected with acute rheumatism also showed evidence of infection. Furthermore, "8.66 per cent. of the siblings of these children were also infected. Simultaneous infection of several members of the same family was frequently noted."

These observations have practical importance. They emphasize the infectiousness of rheumatic fever and point to at least one way by which its occurrence may be prevented. The fact that lowered resistance plays an important rôle is also worthy of thought. The younger the individual infected with rheumatic fever, the greater the likelihood of cardiac involvement and subsequent damage.

The evidences of endocarditis often manifest themselves during the third week of rheumatic fever, heralded by an increase of fever without renewed arthritis with acceleration of the pulse, precordial pain, or hyperesthesia. Prolongation of the a-v conduction time is demonstrable with considerable frequency. The latter together with increase in the size of the heart when present indicates myocarditis, an accompanying condition more frequently present than absent when the endocardium is inflamed. Pericardial involvement is frequent and readily overlooked or mistaken for cardiac enlargement. Sudden changes in heart size are usually pericardial in origin.

Systolic murmurs are diagnostically important if they develop or change in character under observation; also if they are widely transmitted and accompanied by greater loudness of the pulmonic second sound than is to be expected considering the age and position of the patient. Presystolic and diastolic murmurs, although far more important as evidences of valvular disease, rarely appear during the acute stage of the initial attack. If present in marked degree one always suspects an antedecent attack of endocarditis. Precordial consciousness, slight pain, or hyperesthesia may be observed. Tenderness to percussion, especially in the apical region, is occasionally noted. Slight and often brief recurrences of fever after the subsidence of joint

symptoms are always very suggestive of cardiac involvement, especially if associated with an increased pulse rate, persistent anemia, and failure to gain weight or strength.

Arthritic symptoms, sometimes absent in children, are also often slight in adults. This seems to be strikingly the case in China where mitral stenosis is common and rheumatic arthritis rarely observed.[1]

"Subcutaneous nodules" are diagnostically important. These structures vary in size from a millimeter to a centimeter. Often symmetric in distribution they occur along the tendons especially at their insertion and in the neighborhood of the joints. They sometimes occur over muscular tissue. They are hard, generally painless, usually movable, and last from weeks to months. They should be sought for over the elbows, knees, wrists, hands, ankles, head, hips, clavicle, and scapula. They are often undetected because unlooked for, but, judging from description, seem more common in England than with us. According to Coombs[2] they are very similar in structure to Aschoff bodies—"the subcutaneous nodule is to the cardiac nodule, as the tuberculous mass to the miliary tubercle." In adults subcutaneous nodules may be due to other causes, but in children such lesions are almost surely rheumatic.[3] That we are probably remiss in our lack of search for nodules is indicated by the fact that Coates was able to find them in 18 out of 23 rheumatic children; in 6 cases without murmurs and in 5 before any evidence of heart involvement.[4]

Leukocytosis of minor degree is usually demonstrable. Antirheumatic drugs may inhibit an increase in the leukocyte count. Individuals with polyarthritis and hyperpyrexia show higher counts than those with extensive proliferative lesions. Re-

[1] Meleney, H. E., and Rellen, I.: Mitral Stenosis with Rheumatic Fever in North China, Arch. Int. Med., 1924, xl, 525.

[2] Coombs, C.: Jour. Path. and Bact., 1910, xv, 489.

[3] Denzer, B. S.: Rheumatic Heart Disease in Children Under Two Years of Age, Jour. Amer. Med. Assoc., 1924, lxxxii, 1243.

[4] Coates, V.: Rheumatic Infection in Childhood; The Subcutaneous Fibroid Nodules as an Early Manifestation, Br. Med. Jour., 1925, i, 550.

peated leukocyte counts are a useful method of detecting the persistence of infection.[1]

Treatment.—Complete prolonged rest in bed during the attack and a similar régime for weeks or months during and after convalescence are the most important factors. The salicylates relieve pain. They neither decrease nor increase the likelihood of cardiac damage. During convalescence any measures which do not interfere with rest and which may increase the patient's resistance may be employed. It will be interesting to see if the roborant effects of heliotherapy yield as good results as they do in the bone and joint tuberculosis of childhood. The most difficult question to decide is that of focal infection. Is the tonsil in a given case really the source of infection, and if so when is removal advisable? Aside from the operative risk—chiefly pulmonary abscess—we have all seen endocarditis reactivated by tonsillectomy. No operation is justifiable for weeks after all evidences of activity have disappeared. It is better to run the risk of waiting too long than that of operating too soon. In order to prevent later recurrences, removal of all foci infection, the avoidance of exposure, bed rest for every acute cold should be enforced.

Even a complete and successful tonsillectomy by no means insures freedom from future rheumatic infection. Almost as many tonsillectomized children develop rheumatism as do the non-operated group. During convalescence patients should be carefully observed. Temperatures should be taken twice daily at the same hour—preferably the late afternoon, the thermometer to be retained for several minutes before recording the reading. The patient is allowed out of bed only if the rectal temperature remains below 100° F., if leukocytes are under 9000, if he is gaining weight, and is free from symptoms.

The multiplicity of new drugs advocated bespeaks their lack of effect. The salicylates in adequate dosage still hold first place as the best analgesic remedy. To be efficacious salicylates must be administered in large dosage. Experiments upon dogs in-

[1] Swift, H. F., Miller, C. P., and Boots, R. H.: Leukocyte Curve as an Index of the Infection in Rheumatic Fever, Jour. Clin. Investig., 1925, cxcvii.

dicate that to produce cardiac damage more salicylates must be given than is clinically possible.[1] The salicylates perhaps lessen exudative lesions somewhat, but they exercise no effect upon proliferative changes. The injection of silver salts, cincophen, and other medicinal substances fail to yield adequate results. Blister autoserum has been recommended,[2] as has also rheumatic streptococcic vaccine.[3]

No protein therapy, non-specific or otherwise, should be undertaken without due consideration inasmuch as these procedures may reactivate valvular infection to a clinically recognizable degree.[4]

SUBACUTE BACTERIAL ENDOCARDITIS

(Endocarditis lenta, non-hemolytic streptococcus endocarditis)

This disease is often overlooked because it masquerades in many guises in which symptoms pointing to the heart may be slight or entirely lacking. It is essentially a low-grade non-hemolytic streptococcus infection of the endocardium, for our knowledge of which we owe much to Emanuel Libman. A small group of cases are due to the influenza bacillus. Secondary infection with other organisms such as the pneumococcus may occur.

In rheumatic fever the heart suffers from a primary focus elsewhere. In subacute bacterial endocarditis the heart represents the primary focus, and thrombotic manifestations occur elsewhere.

Pathology.—The mitral valve is involved in about two-thirds and the aortic in one-third of the cases. The vegetations often large, especially in case of the aortic valve. Their color varies—pinkish, reddish, yellowish, or greenish. Very often the mural endocardium and the chordæ tendineæ are extremely involved.

[1] Ribiere, P., and Pachon, E.: Chronic Rheumatic Heart Disease, Paris Med., 1924, 2019.

[2] Iacchia, P.: Autoserotherapy by Cantharidal Blister Fluid, Pediatria, 1925, lxxxix.

[3] Gregory, H. H. C.: Vaccine Treatment of Acute Rheumatism, Brit. Jour. Child. Dis., 1924, xii, 422.

[4] Spangler, G.: Focal Reaction on Heart Valves, Wien. Klin. Woch., 1924, 1325.

Embolization is frequent and accountable for some of the most striking clinical features. Bacterial (mycotic) aneurysms occur more frequently in this than in any other type of infection.[1]

Symptoms.—These are aptly placed by Libman in three groups. First: Symptoms which are due to toxemia and the anemia resulting from it. Second: Symptoms which are due to pieces breaking off from the valve—these we call endocarditic symptoms. Third: There may be symptoms from myocardial weakness. It is important to remember that the cardiac symptoms are very often not marked in cases of this disease. In a large number of the cases in which cardiac symptoms develop they are due to weakness of the heart muscle from anemia and toxemia rather than valvular defects as such.

The onset is often insidious and without definite symptoms. Sometimes, however, the initial symptoms are definitely referred to the heart, as, for instance, shortness of breath on exertion, palpitation, and cough which is sometimes attended with blood-streaked sputum. Fever is present at times in all cases. It is often slight, but in the majority of cases is high and intermittent in the late stages of the disease. If splenic infarcts occur, the fever is apt to be high and intermittent. Chills and sweating may occur in association with the fever. Loss of weight and extreme weakness are noted in most cases.

Precordial pain or a sense of oppression in the chest is of frequent occurrence. Pain in the joints is not uncommon and may be associated with slight transitory swellings. A purpuric eruption may be present also. Libman has called attention to an important diagnostic sign, namely, tenderness over the lower part of the sternum. The lightest tap with the percussing finger may cause marked pain.

Physical Examination.—An important diagnostic feature is the presence of small, painful erythematous nodules in the skin of the hands and feet. These cutaneous nodes rarely occur except in this form of endocarditis. If carefully sought they will

[1] For complete recent study see Stengel, A., and Wolferth, C. C.: Mycotic (Bacterial) Aneurysms of Intravascular Origin, Arch. Int. Med., 1923, xxxi, 527.

be found in about 40 per cent. of the cases (Blumer). They are due to minute emboli in terminal superficial vessels. Petechiæ in the skin and conjunctival mucous membrane are frequently noted. Minute linear hemorrhages may be seen around (Horder) or under (Blumer[1]) the finger-nails. Curvature if not actual clubbing of the finger-nails also occurs. Indeed Cotton found it in 70 per cent. of his cases. It did not seem dependent upon cyanosis. It occurred with both mitral and aortic involvement and regardless of whether rheumatic or syphilitic infection preceded it.

The heart examination yields variable but in no wise characteristic results. An old murmur may become modified. New murmurs may appear, or no murmur may be audible. More or less enlargement is usually demonstrable; pericarditis is rare. In this respect subacute bacterial endocarditis differs strikingly from rheumatic infection.

The leukocytes are often increased in number. A characteristic feature is the presence of a more or less marked anemia, which, in many cases, is progressive. Libman has emphasized the peculiar sallow or waxy color of the face in these cases. Later the face may assume a coffee-colored tint. In cases which become bacteria-free, the face and other portions of the body often assume a diffuse brown color. The spleen is enlarged in nearly all cases and is easily felt on palpation. In some instances the organ is enormously enlarged as a result of infarction. Many cases masquerade as seemingly causeless anemias which respond to no treatment and become progressively worse.

A striking and diagnostically important finding is hematuria. This may be obvious upon inspection if renal infarction has occurred. Diffuse hemorrhagic glomerular nephritis is very common, but often a microscopic search for red corpuscles may be necessary. Cultures from the urine may yield the offending micro-organism.

A positive diagnosis can be established by means of blood-cultures. Repeated attempts are, however, often necessary and

[1] Blumer, G.: Digital Manipulations of Subacute Bacterial Endocarditis, Amer. Heart Jour., 1926, i, 257.

the established technic must be carefully followed. One should remember, however, that periods exist in which no bacteria can be demonstrated in the blood-stream, although active infection of the endocardium persists. These so-called bacteria free stages result either from the fact that the few organisms which are thrown off into the blood-stream are promptly killed or to the fact that they remain deep-seated and surrounded by inflammatory reaction.

Termination.—Death usually occurs within four to eighteen months. Some cases are more chronic and a few spontaneous recoveries have been reported.[1] Death generally results from embolic causes, or from gradual heart failure. Occasionally an embolic aneurysm leads to a rapid ending.

Treatment.—No specific form of treatment has any value. The blood-stream does not appear to lack bactericidal substances, but apparently the organisms are too deeply located to be accessible to it. Rest in bed coupled with measures tending to increase general resistance is about all that can be accomplished in so far as recovery is concerned.

ACUTE BACTERIAL ENDOCARDITIS

This form of endocarditis is a secondary manifestation of active purulent infection elsewhere. It may be caused by many different organisms, though most frequently the etiologic factors are hemolytic streptococci and staphylococci.

One should remember, however, that micro-organisms undergo changes in the body. A local pneumococcus infection may in the blood-stream approach a non-hemolytic streptococcus. Libman[2] believes that non-hemolytic streptococci represents transition forms between pneumococci and hemolytic streptococci.

Clinically, acute endocarditis occurs not infrequently as a complication of pneumonia and gonorrhea. Rarely it appears in tuberculosis, typhoid fever, scarlet fever, diphtheria, variola, dysentery, etc.

[1] Libman, E.: Consideration of Prognosis in Subacute Bacterial Endocarditis, Amer. Heart Jour., 1925, i, 1.

[2] Ibid.: Observation on General Infection by Bacteria, Jour. Mich. State Med. Soc., 1924, November.

PNEUMOCOCCUS ENDOCARDITIS

Pneumococcus endocarditis is generally characterized by the severity of the local lesions and by frequent involvement of the aortic orifice. Rarely it may occur without a demonstrable preceding pneumonia. It is said to be more frequent in children than in adults. It is sometimes part of pancarditis.

The symptoms vary somewhat and hence a number of types are recognized, the septic, the typhoid, the cardiac, the embolic, and the cerebral.

Among 32,349 cases of lobar pneumonia it was noted clinically in 144 (0.44 per cent.). Among 2639 fatal pneumonias autopsied it occurred in 157 instances (5.8 per cent.).[1]

GONORRHEAL ENDOCARDITIS

Gonorrheal endocarditis as we know it is a malignant vegetative and ulcerative infection not unlike that produced by the pneumococcus. The vegetations are large, often penduculated. Extreme ulceration and even myocardial abscesses may occur. The gonococcus has been demonstrated in pure culture from some cases. The septicemia of gonorrhea is next to that of the puerperal state, and pneumococcus infection the most frequently accompanied by septic endocarditis (Vaquez). The aortic and pulmonary valves are most frequently involved. Infection of the endocardium may occur as early as the fifth day and as late as the eighth month in a case of urethritis. It is more common in men, owing to the greater frequency of cystitis and prostatitis. In a few cases the gonococcus has been cultured from the blood-stream. Among 176 cases of endocarditis of determined origin 20 (11.3 per cent.) were gonococcal. The organisms were collected during life in 10 and postmortems in 14 instances. (Thayer[2]).

Gonorrheal endocarditis usually begins suddenly with chills, high fever, leukocytosis, anemia, and severe constitutional symptoms. Healthy valves are often infected, pericarditis and myo-

[1] Norris, G. W.: Croupous Pneumonia, Oslers Mod., Med., 1925, i, 235.

[2] Thayer, W. S.: Cardiac Complications of Gonorrhea, Trans. Assoc. Amer. Phys., 1922, xxxvii, 248.

carditis are common. Mural endocarditis is not unusual. The disease, which is probably commoner than is generally supposed, usually ends fatally in from four to nine weeks.

Tuberculous endocarditis is an autopsy curiosity not a clinical recognizable condition. Tubercle bacilli have occasionally been demonstrated. More frequently a characteristic tuberculous histology has been found. More or less simple fibrous changes have been ascribed both to the bacillus and to its toxin, often without sufficient justification.

DIAGNOSIS OF HEART FAILURE, ACUTE AND CHRONIC

CHARLES C. WOLFERTH

IN the entire range of clinical medicine there is no subject more important nor of more universal interest than the one your Committee has done me the honor of assigning for today's seminar. It touches practically every field of medicine. It comes under consideration in all illnesses that may prove fatal. It concerns the surgeon as well as the physician.

Consideration of the subject of heart failure naturally falls into two main divisions, namely, the physiologic and the clinical aspects. Concerning the former, our knowledge is far from satisfactory. It is a subject tremendously difficult of investigation, and further notable progress will probably have to await advances in our knowledge of muscular contraction, particularly cardiac muscle contraction.

At present it is frequently impossible to advance a reasonable explanation for cardiac failure even after careful anatomic study of the tissues involved. On the clinical side, however, which is our field of discussion for today, we are in a much better position. The clinical evidences of heart failure may usually be discovered by any physician who looks carefully enough for its signs. It is the exception rather than the rule that knowledge now available does not make possible a reasonably accurate evaluation of these signs.

Before entering into a discussion of heart failure it is advisable to state what we mean by heart failure. At best it is a relative term and scarcely susceptible of exact definition. Some writers use it as synonymous with cardiac decompensation, and go as far as to recommend the abolition of the latter from our nomenclature. It is preferable to designate as decompensation the state of heart failure with the patient at rest, employing the

term heart failure to cover a broader field. For the present discussion it may be defined loosely as inability of the heart to fulfil efficiently the demands made on it. This definition would, therefore, include the concept of a relatively healthy heart which had failed to perform efficiently because of excessive demands for work. Thus, if an untrained man ran a 440-yard race he might show extreme breathlessness, cyanosis, and acute cardiac dilatation—unmistakable evidences of heart failure according to our definition—yet if he had been properly trained his physical activities would be limited by skeletal muscle fatigue rather than heart failure.

It is necessary for the clinician to make a distinction between heart failure and circulatory failure. It is true enough that circulatory failure is most often due principally to heart failure, but other important factors are frequently concerned, namely, the blood-vessels, the vasomotor mechanism, the lungs, and the state of the blood. In the presence of circulatory failure, it is not to be assumed that the fault lies entirely with the heart until other possible contributory factors have been carefully investigated and their efforts considered. These problems are rendered more difficult by the fact that the circulation is maintained by a complex system of delicate checks and balances—faulty action in one part tending to be compensated by overaction of others.

If we are to recognize heart failure, particularly in its minor manifestations, it is necessary to be familiar with its clinical causes. The most important cause by far is active infection—either actual infection of the heart or infections elsewhere exerting toxic effects on the heart. Severe general infections may cause the heart to fail quickly. Attention should also be called here to the end-results of infection of the heart by the rheumatic virus, bacteria, or syphilis, which by crippling the pericardium, muscle, or the valves, impair the vigor of its contractile power or impose mechanical handicaps tending to nullify the best efforts of the muscle.

Next to infections in importance are the so-called degenerative diseases—chronic myocardial disease with sclerotic or fatty changes, arteriosclerosis, and glomerular lesions of the kidneys.

Evidence is accumulating that infection is also important in the production of these degenerative diseases.

Other important contributory causes of heart failure are hypertension, obesity, prolonged nervous or physical strain, diseases of the thyroid gland, diseases of the lungs, such as asthma and pneumoconiosis, anemia, and congenital cardiac deformities. A host of other conditions of less importance might be mentioned. One extremely important group of causes concerned in the production of heart failure desiring special consideration not only because of its frequency, but because it so often tips the balance which the heart has been struggling to maintain, comprises the abnormal rates and rhythms. These, however, are secondary not primary causes and may be referred back either to a functional aberration of the heart muscle or its nervous control, or both. The truth of this statement is obvious enough, but is frequently insufficiently emphasized in writings on the subject of abnormal cardiac mechanisms. Attention is too frequently focused on arhythmia and not the cause of arhythmia.

Methods.—The tremendous interest manifested in the subject of heart disease during the past twenty-five years has stimulated a great volume of research. This, in turn, has led to attempts at clinical application of a great variety of methods to discover the functional efficiency of the heart. As might be expected, many of these have fallen by the wayside and others are in the process of falling. Some have real value, but it should be strongly emphasized that special tests occupy but a subsidiary place in the study of cardiac function.

The history easily takes first place in importance. We are entirely dependent on it for the subjective sensations of the patient which are usually prominent in heart disease. In spite of the frequency of reports of sudden death without premonitory symptoms, it is rare that symptoms cannot be elicited by proper questioning in the presence of severe heart disease. One should inquire particularly as to the effort tolerance, precordial or substernal pains, palpitation and fatiguability, and such minor grades of edema as tightness of the shoes toward evening. A history of repeated bronchitis or bronchopneumonia, chronic

cough, or asthmatic attacks, digestive symptoms of various kinds, so-called neuralgia, neuritis, or rheumatism in the distribution of the lower branch of the trigeminal nerve, the lower cervical and the thoracic segments, various cerebral symptoms, such as vertigo, loss of consciousness, mental dulness, or even psychoses, and many other less important symptoms may be cardiac in origin.

It is obvious that a diagnosis is not usually possible on history alone, yet the only physical signs from which positive proof of heart failure may be obtained are the so-called congestive phenomena and to some extent the behavior of the pulse. Even in the presence of these it is sometimes necessary to consider other factors beside the heart itself, which may be contributing to the circulatory breakdown. As Mackenzie has pointed out, one obtains no direct evidence of heart failure from the examination of the heart itself; nevertheless, unmistakable signs of serious heart disease may be discovered which in the presence of other appropriate phenomena offer strong presumptive evidence of heart failure. Many clinicians profess to be able to detect appreciable differences in the size of the cardiac outline in their patients from day to day as they improve or grow worse. There is no doubt that in some cases of acute failure the heart may dilate greatly and with improvement diminish correspondingly. *x*-Ray evidence would seem to indicate that changes great enough to be readily detectable clinically are not usual. We are forced to the conclusion that those who find these changes in so many cases are frequently deluding themselves.

If we had an ideal functional test of the heart, many of our diagnostic problems would be simplified. Response to various forms of physical exercise have been carefully studied. Of the tests thus far devised, probably the best is stair-climbing at prescribed rate. At most, this is a test of physical fitness of which the state of the heart is only one factor. In youthful patients it may yield information of undoubted value as to the functional state of the heart, but in the elderly it is practically useless.

The sphygmomanometer furnishes one valuable bit of evidence of a failing heart, this being a narrowing pulse pressure.

Due allowance must be made for variations in the cardiac rate and the height of systolic pressure. Unfortunately, we have no accurate formulæ to correct for these variations so only gross changes in pulse pressure may be regarded as of significance. Nevertheless, a narrow pulse pressure is usually excellent evidence of inefficient action of the heart.

The sphygmomanometer offers also the most delicate method we have of detecting continuous pulsus alternans. This disturbance of cardiac action, provided the rate is not rapid, points strongly to failure or at least impending failure of the heart.

Determination of vital capacity has been proposed as a test of the functional state of the heart. If cardiac failure is present, this method has a certain value as a quantitative measure of change in the degree of failure. It has not yet been demonstrated to be of much utility in discovering the presence of cardiac insufficiency not obvious by ordinary methods of examination. Possibly the unsatisfactory state of standards will not permit the test to have much diagnostic importance in heart disease, valuable though it is in the study of the lungs.

The electrocardiograph like direct physical examination of the heart offers only presumptive evidence of failure. Nevertheless, not infrequently, the electrocardiograms reveal the presence of serious myocardial damage which is not discoverable by our present methods of physical examination. As our experience with electrocardiography grows, it is found an increasingly valuable method of study of the condition of the heart muscle. The method is comparatively new. The workers in this field have been fascinated by the pathologic results and have thus far almost neglected the unexciting and laborious task of collecting sufficient data on normals and classifying it so that the significance of what are now regarded as minor abnormalities may be properly evaluated.

***x*-Ray.**—Some observers claim to be able to detect under the fluoroscope degrees of vigor of cardiac contraction that enables them to form a judgment as to the capacity of the heart. Such a view is not generally held at present.

The chief value of the *x*-ray is to give us an accurate sil-

houette of the heart. Gross differences from the normal are readily detected, but here also, as in the case of electrocardiography, standards of what is normal are inadequate. When these are established, *x*-ray will probably become a more valuable method of study than it is today.

The statement is frequently and very properly made that cardiac enlargement is one of the most reliable evidences of heart disease. On the other hand, it cannot be too strongly emphasized that the presence of a heart regarded as well within the normal limits of size by no means rules out the possibility of disease or failure. Patients of middle age or beyond frequently suffer severe failure, although the heart remains within normal limits of size.

Acute Heart Failure.—There are two common causes of acute heart failure, namely, chronic heart disease and severe acute infection. The latter may attack the heart directly in which case its effects are especially disastrous. More commonly it is what we call the toxemia of infection that attacks heart muscle-fibers and causes disintegrative changes that are readily recognized by the pathologist. It is not difficult to understand why hearts so affected are unable to carry on their work.

In the presence of chronic heart disease there is usually some precipitating cause of acute failure. This may be moderate over-exertion, overloading the stomach, a mild infection, or a hundred other things. Patients are frequently inclined to attribute their trouble entirely to the precipitating cause. The more vulnerable the heart the more readily it is upset by trivial disturbances. It is therefore a safe inference that the heart is seriously diseased if an overloaded stomach or any other minor handicap causes acute failure. It is difficult, if not impossible, to cause more than transitory failure of a healthy heart by any method except poisoning.

The clinical picture of acute heart failure is attended with such spectacular phenomena that there is usually little difficulty in recognizing that something is seriously wrong with the circulation. The types which the examiner is most likely to overlook are those with conspicuous cerebral phenomena, or those in

which pains or distress outside the usual cardiac distribution direct attention away from the heart. Such mistakes will rarely occur if the ordinary methods of eliciting history and physical examination are not neglected.

It is frequently difficult, however, and sometimes impossible to tell at which end of the circulatory apparatus failure has occurred, the cardiac or the peripheral vasomotor. What advances have been made in this field we owe almost entirely to the physiologists. They have taught us that the picture of shock—leaky skin, extremely rapid, small, soft pulse, low blood-pressure, ashy gray cyanosis, weak, almost imperceptible heart sounds—is not due to heart failure, but peripheral vasomotor failure. The circulatory failure of severe acute infection is apparently a mixture of cardiac failure and peripheral vasomotor failure; frequently the latter is the more important. The truth of this statement can be readily established by observing the effects of blood transfusion in the terminal stages of pneumonia after circulatory collapse has become severe. The extremely rapid, almost imperceptible pulse may again become much stronger and less rapid and the general state of the circulation improve remarkably for a period of hours.

Valuable signs of acute heart failure are rapid dilatation of the heart, engorgement of the veins, and the development of congestive phenomena. Heart failure is possible without any of these. There may be only enfeeblement and perhaps even slowing of the heart beat. The symptoms of this type are mainly cerebral due to insufficient supply of blood.

Chronic Heart Failure.—There is no clinical picture more familiar than that of chronic heart failure and it need not be described here. Yet the symptoms may be atypical and thus escape recognition, or as is not infrequently the case there may be complicating factors that modify the picture.

In the first place, it is necessary to distinguish between actual heart failure and conditions simulating it. Before the World War one commonly encountered cases that were diagnosed irritable heart or nervous heart. During the war these conditions were tremendously exaggerated and received such names as

"effort syndrome" or "neurocirculatory asthenia." Careful study proved that they were not due to heart disease, although their actual causes are still unknown. The clinical pictures of effort syndrome now encountered, with the exception of those found in cardiac and nervous clinics of the Veterans' Bureau are identical with those seen before the war and may easily be mistaken for heart disease if not carefully studied.

Precordial pain or distress is one of the commonest symptoms found among those with either real or fancied heart disease. It should never be dismissed lightly as it is frequently the first subjective evidence of a failing heart. This is particularly true of substernal pain or pain on effort. The more or less continuous aches or pains uninfluenced by exertion and centered about the apical region do not necessarily indicate heart disease. They may be merely an evidence of cardiac fatigue or irritation. The precordium is also a favorite site for pains of psychoneurotic origin.

Shortness of breath is the most reliable and generally satisfactory evidence of heart failure we possess; yet like almost all other symptoms it may be due to a wide variety of causes, such as neurosis, pulmonary disease, anemia, or acidosis. Nervous breathlessness which frequently comes under consideration in relation to heart disease is liable to be bizarre in its manifestations with attacks of unaccountable shallow tachypnea or single long deep breaths at irregular intervals. The condition is often alarming to patients suggesting to them the presence of heart disease.

Attention should be also called to the fact that acrocyanosis is not necessarily an evidence of heart disease. It is found in certain peripheral vasomotor disturbances, notably the effort syndrome. Cyanosis due to heart disease tends to be general in its distribution and involves the mucous membranes.

The physiologic relationship between the heart and lungs is so close that heart failure nearly always causes pulmonary disturbance. This may vary in degree from slight congestion to tremendous pulmonary edema or massive hydrothorax. Thus, a variety of pulmonary disorders may be simulated by heart disease. Per-

haps the most frequent of these is chronic or recurrent bronchitis. Furthermore, congestion of the lungs does predispose to bronchitis or bronchopneumonia so that attention may be centered on the lungs where the heart is fundamentally at fault.

Cardiac asthma has been mentioned so much that many have the impression that it must be a common condition. On the contrary it is quite rare, so that in the presence of both severe asthma and heart failure it is far more likely that the cardiac disturbance is due to the asthma than otherwise.

The presence of cough, or hemoptysis, may lead to the suspicion of pulmonary tuberculosis. This is true particularly in mitral stenosis in which blood spitting occurs at an earlier stage of failure than in any other form of heart disease. Mitral stenosis may also cause impairment to percussion at the left apex anteriorly which may be mistakenly regarded as evidence of a tuberculous lesion.

The interrelationships of cardiac and digestive symptoms are so well known that it is scarcely necessary to mention them. Cardiac symptoms of digestive origin may be and frequently are trivial and unimportant, but digestive derangements of cardiac origin are practically always evidences of heart failure. The liver disturbances may be particularly misleading, causing mistaken diagnoses of gall-bladder disease or various diseases of the liver itself. The pain of congestion may occasionally be intense, and those not familiar with it may be driven to operate with the expectation of finding gall-bladder disease. On the other hand, congestion of the liver is frequently entirely painless and may escape observation until a well-marked cardiac cirrhosis with portal obstruction is produced; under such circumstances, tuberculous peritonitis may be considered in youthful patients, and Laennec's cirrhosis or malignancy in those who are older.

The recent attempts to relieve the pains of angina pectoris by surgery have focused attention sharply on the subject of pains of cardiac origin. Ordinarily, these offer no diagnostic difficulties, at least so far as their source is concerned. When they occur in unusual distributions, such as the trigeminal nerve, in the epigastrium or in localized areas of the shoulders,

arms, or fingers, their significance is readily missed if the possibility of their being cardiac in origin is overlooked. Spondylitis, root pains, neuritis, or neuralgia may be mistaken for angina. This is not so remarkable if we recall that the same sensory pathways serve for all.

The cerebral manifestations of cardiac failure are varied. The psychoses may manifest themselves as states of depression or the wildest excitation. These occur, as a rule, in well-advanced heart failure, but the connection between the mental state and the heart may remain unsuspected. Milder forms of heart disease are liable to cause emotional upsets or changes in personality. The cerebral seizures of Stokes-Adams' syndrome have not only been known since the description of these authors, but had previously been reported by Morgagni. Milder cerebral seizures, epileptiform in type, are seen particularly in patients with slow pulse and low blood-pressure.

PERICARDITIS

WILLIAM E. HUGHES

IN speaking of this subject I am afraid I cannot bring out anything new, because there is nothing new; there is really nothing new in any of the ordinary diseases. It is simply a matter of going over the subject and of recalling your attention to a number of little details which you might possibly have forgotten.

Pericarditis is an inflammation of the pericardium; it is like an inflammation of the serous membrane, secondary in the same way to some other disease or condition. It is not a primary affair. It is not idiopathic; that is, in the strict sense of the word there is no such thing as idiopathic inflammation. Inflammation has a definite bacterial cause. In that sense you can say that pericarditis is always secondary; it is not only secondary, but it generally occurs in the course of its provocative cause; the disease either introduces the germ into the pericardium or heart in a general way through the blood-stream, or it so lowers the resistance that germs are permitted to produce a specific effect on some of the serous membranes or these germs may be introduced by direct contact with neighboring organs. Possibly you may have a traumatic pericarditis; that also is secondary. I dwell on this somewhat at length, but not needlessly because pericarditis is a condition which has few symptoms in itself; you might say almost no symptoms; it is diagnosed by physical signs rather than symptoms as I will point out and as you probably already know. Occurring in the course of other affections, it aggravates them and lends a certain additional gravity to them, and if you don't understand clearly the possibility of the occurrence of pericarditis you overlook it and may possibly do your patient a very decided injury.

It is secondary most frequently to rheumatism or to rheumatic condition or what we used to call rheumatism, but what is called arthritis today. You know what I mean when I speak of rheumatism, but that coupled with tonsillitis is the commonest cause of the occurrence of pericarditis. The direct introduction is by means of the blood-stream carrying the germ into the pericardium. Why is it lodged in the pericardium and not in some other serous membrane is something which we cannot explain. There is probably the question of individual susceptibility—one person will develop one type of inflammation, another will develop another type.

Another condition which is complicated by pericarditis is Bright's disease, and there it is probable that the Bright's disease so lowers the general tone of the system as to permit the germs to become pathognomonic; so not infrequently in the course of Bright's disease you have pericarditis. Another disease is pneumonia; there the germ is transferred directly by contact with the diseased lung or else it is a part in a general pneumococcic infection. You can see why it is necessary to bear all these points in mind.

While pericarditis produces symptoms they are not distinctly pathognomonic, merely suggestive. It exaggerates the ordinary condition and symptoms of a disease. In a pneumonia case, if the fever runs up a little higher than usual and the patient does not do so well; if you find a pericardial inflammation you know why these symptoms are present. If a patient with rheumatism gets a high temperature and delirium, you will sometimes find on examination that the real reason for it is a pericarditis. The acute infectious diseases are causative; typhoid fever occasionally, scarlet fever is moderately commonly a cause. Measles, etc., and all that class of diseases may be causative.

Acute pericarditis is not infrequently associated with acute endocarditis, and of course it complicates the heart distinctly unfavorably. There are probably more complicated cases than there are cases of pericarditis without any such association. The influence of the pericardial inflammation on the integrity of the heart is probably not an exceedingly important one,

per se; i. e., you may have a very decided inflammation of the membrane and yet the heart is doing its work pretty well. But rather frequently there is an association of the myocardial degeneration and the pericardial inflammation, and if this is so you have a state of affairs which will produce decided impairment of the heart power.

As far as the pathologic lesion in pericarditis is concerned, it is just as it would be in any other serous membrane—a little roughening, some congestion, and then the throwing off of lymph and probably the effusion of serum. During cure the reverse process is obtained: the absorption of the serum, the relief of the congestion, and the probable organization of lymph and return to the more or less normal heart. I think it is important to recognize the fact that pericarditis without inflammation of the endocardium is of itself an unimportant affair, but where you get implication of the myocardium you get what may be a decidedly serious event. As long as there is simply pericarditis there is decidedly little danger in the condition of the heart, unless decidedly serious degeneration is present. When the serum is profuse you get the mechanical effect on the heart muscles and this is a different problem, although even this need not be a serious affair because the pericardial sac is very elastic and it expands almost indefinitely. After all, the seriousness of the disease goes back to the implication of the myocardium. As far as the symptoms of pericarditis, little disturbance in the general condition of the patient is the most important. If it is one in which the poison has gained access through the tonsils without any other disease, there will be a little fever, general malaise, and a little discomfort. As far as the local symptoms go, the most common one is pain. In my personal experience pain has not been a very important symptom. Certainly, it is not important enough for diagnostic purposes. There is vague discomfort in the precordium. Any discomfort will lead to an investigation and I might say that it is a wise thing in any acute disease to go over the heart and lungs at every visit. It doesn't take longer than a few minutes and may save you a good deal of trouble and anxiety later. Pain is sometimes very marked in the

precordial region, in the left shoulder and down the arms; it resembles angina pectoris, but there is no history of angina pectoris; it is an acute condition. Sometimes pain is evidenced at the epigastrium and not the precordium. Pain is an important symptom if it attracts your attention to the possibility of pericarditis, but not in any pathognomonic way. The patient is more or less uncomfortable; there is a little sense of dyspnea; the temperature is higher than if no complication is present.

There is one symptom which occurs that might be of importance; in quite a few cases of pericarditis there has been pain on swallowing, *i. e.*, pain when the food goes down the esophagus. I regard this as being of some degree of importance. When you have effusion the mechanical element is present and you have all the signs of a rather poor heart action. Dyspnea is more or less decided; patient cannot be comfortable unless he is sitting up; he is cyanosed and the veins in the neck are turgid. There is cough and a little dizziness and sometimes a false delirium. These symptoms all might mean anything; there is nothing about any of them which is definite. It is the physical signs which are diagnostic. In the beginning we get a friction sound and this is the first positive evidence of pericarditis. When it starts I do not know; it may be in the initial roughening of the pericardium; it may be the effusion of lymph; like the pleurisy friction sound it is generally double—to and fro—diastolic being just like the systolic. But it is not necessarily synchronous with the systolic or diastolic; sometimes you get three sounds and sometimes four. You get a sort of a gallop rhythm occasionally. This friction sound is characteristic of pericarditis within certain limits; it most frequently is heard in the third or fourth interspaces over a comparatively limited area, but it may occur at any other part of the precordium. In other cases it is heard all over the precordium, or even all over the left chest and sometimes into the right chest, always with this peculiar scraping sort of a sound. But usually it is limited to some part of the precordium and not conducted beyond it. This friction sound lasts until the effusion occurs; first it disappears over the body of the heart and eventu-

ally over the base. It may persist up to the point where the effusion has become marked. On inspection at the beginning of the dry state there is nothing but perhaps a violent action of the heart. Palpitation will show you a friction fremitus sometimes. Sometimes you can get this with the finger tips; sometimes the whole hand is necessary. Percussion shows nothing extraordinary before the effusion in the size of the heart. The next stage is effusion. The physical signs are definite, but hard to elicit until the effusion is advanced. Thus on inspection you find possibly bulging in the region of the precordium. On palpation the apex-beat of the heart and the impulse of the heart slowly disappear. The apex-beat at first rises and then disappears. The impulse of the heart grows weaker; it may be undulatory; it also disappears. On percussion you get quite characteristic physical signs and a decided area of dulness which is described as pear shaped, that is triangular. It has a broad base and extends beyond the apex-beat. This is important diagnostically. An area of dulness extending beyond the apex-beat is pretty sure to be pericardial effusion. Of course we must remember that the apex-beat is going to disappear shortly. Another type is an area of dulness to the right of the precordium, and if you can demonstrate this you can be pretty sure of effusion. Auscultation simply shows the heart sounds getting fainter and fainter; they disappear over the apex-beat, and then over the body of the heart and then over the base. As the effusion subsides you get a return of the friction sound and that eventually fades out when your patient has recovered. At the time of the largest effusion there is another physical sign which is of importance and which may mix you up; that is, an area of dulness in the back about the angle of the scapula and with it tubular breathing. This tends to make us diagnose pneumonia, which is a wrong diagnosis. If you get your patient to lean forward, the area of dulness will grow less or disappear—this will be of diagnostic importance. Then, around the area of effusion there is rather likely to be a zone of high-pitched tympany due to compression of the lungs. Another point is that full inspiration does not change the area of dulness nearly as much as it

does in normal cardiac dulness. The lung does not come as far over the area of effusion as it does over the normal heart.

Differential Diagnosis.—In the beginning in the stage of friction you are not likely to mistake it for any cardiac murmur. It is double; it is very superficial and not infrequently you will get it coming out a little bit plainer with moderate pressure and disappearing with greater pressure. Cardiac murmurs are deeper; you rarely have two which are as nearly alike as the two elements of the friction sound. The aortic double is the one which more nearly approaches it, but that too has its peculiar characteristic. There is sometimes over the body of the heart, exactly in the region of the pericardial friction sounds, a superficial rub; it is systolic and is associated I think with a dilatation of the right side of the heart. You might possibly make a mistake there, but it is not quite like a friction sound without the history of the pericarditis, but with the history of some long-continued failure of the heart power—a single murmur and not distinctly rubbing. You can dispose of this and of the endocardial murmur.

Not infrequently you get a murmur out toward the apex-beat not near the base of the heart or sternum which is synchronous with inspiration and ceases during expiration. There is no rubbing and it is caused by the heart beating against the lungs. Bear this in mind. The thing which is hardest to diagnosticate is where you have pleurisy over the heart; of course if you have pleurisy at any other part of the lung it is easily differentiated by getting the patient to stop breathing when the sound ceases. But you have a different proposition if it is lying over the area of the heart. It is influenced by inspiration, but also by the heart-beat. You can make a differential diagnosis by making the patient hold his breath when the murmur will become single instead of double. Bear in mind, too, that this murmur is well out toward or beyond the apex-beat and is not close to the sternum. So you can make your diagnosis.

In the stage of effusion sometimes there is more doubt. The effusion simulates dilatation of the heart; that is, a distinctly weak heart, that has been going on for some time. If you have

been watching the case you will know that the dilatation has not been preceded by a friction rub, the apex is not raised, it is rather toward the epigastrium; it is over a wide area, there are very distinct evidences of heart weakness. The least little exertion will throw the patient into a state of dyspnea or palpitation (?). The heart sounds are perfectly clear and the first sounds are weak. In pericardial effusion such is not so. The sound is muffled and not distinct in character. In dilatation the area of cardiac dulness is decreased in full inspiration just as the normal heart. Again in dilatation there is not much likelihood of dulness to the right of the sternum, although in the case of extreme dilatation you might find it so. In large amounts of effusion the possibility of dilatation is eliminated because no dilatation could be as great as this is.

I remember 1 case which puzzled me greatly and was only solved at postmortem—it was, apparently, a long-continued pericardial effusion; the apex-beat was fairly clear; there was a somewhat large area of dulness which extended to the left beyond the apex-beat; postmortem revealed an aneurysm of the large left ventricle. In this case it was almost impossible to make a differential diagnosis. Sometimes the pulse is a matter of importance symptomatically or diagnostically. The ordinary pulse in pericardial effusion is rapid, weak, irregular, but sometimes where the effusion is large or where there is adherent pericardium following we get the pulsus paradoxis (the pulse disappearing during inspiration), and another thing which characterizes pericardial adhesions is the emptying of the jugular veins during diastole. Both of these mean pericardial involvement of one kind or another. They are important diagnostic signs, but not in themselves sufficient to designate the disease.

What is the future and what are the dangers?

As I have said in the first stages there is comparatively little danger unless you find a heart weakened by myocarditis.

In the next stage, the stage of effusion, there is danger, but the danger is rather through myocardial degeneration than through pressure of the fluid. Sometimes on aspiration you get a quart or so of fluid out of a distended pericardial sac and there

has been no harm done to the patient. It shows how almost limitless and extensive is the stretching ability of the pericardium. If there is much dyspnea and if there is much cyanosis, if there is much distention of the veins of the neck, and if the heart power is unquestionably failing, you have danger and this danger may be somewhat curtailed by relieving the mechanical pressure of the effusion.

As to the future of these cases: pericarditis does not necessarily subside promptly. It may run an almost indefinitely. The friction sound may come and go. I would say of the friction sound that it is exceedingly irregular; it changes with change of posture, and with changes in pressure, and it changes from hour to hour, much more so than the friction sound of pleurisy.

Quite a number of these cases are tuberculous in origin. Just like tuberculous pleurisy they may eventually recover. Why tuberculosis should attack the pericardium infrequently and the pleura so exceedingly frequently is something which I cannot explain. There is, of course, the question of individual susceptibility.

In any inflammation of the serous membrane you are likely to have adhesions. The pericardium is not exceptional in this rule. These adhesions when the result of ordinary pericarditis are not of very great importance. But where you get it long continued and especially tuberculous, then you get wide-spread inflammation around the pericardium and in the mediastinum and this produces another element of danger. You get a crippled heart; adherent to the anterior chest wall and to the spinal column it does not get any chance to expand. This is the adherent pericardium, with which we meet occasionally, but it is not the ordinary result by any means of pericarditis.

As to **treatment** there is no treatment for pericarditis in itself; when it appears as a rheumatic complication, I use antirheumatic medicine. I still believe in the salicylates in rheumatic conditions, and I would therefore give them in the treatment of pericarditis occurring in the course of a rheumatic condition. Then, of course, you do what you can for the comfort of the patient. I do not advocate blistering, which I gave up

some time ago. Iodin might be used as a counterirritant, or you may use an ice-cap. Keep the patients at rest, although this is not as necessary in pericarditis as in endocarditis. Of course, if you have a weak heart you treat it as you would any other weak heart.

As to duration, as soon as the acuteness is over, you can let the patient go about. I don't think there is much danger in this. Patients with endocarditis should be kept in bed for three or four months. With pericarditis they need only stay in bed a comparatively short time. When it comes to cases of effusion you have a mechanical problem, and just as in any other effusion you use mechanical drainage. I think it is scarcely worth while giving the patient purgatives in order to drain it off, but I do give diuretics and it is better to alkalinize the patient a little. Of course it is said that no one should use a trochar or canula for anything except a surgeon, and that he should use it only preparatory to operation; but in spite of this I still feel that if you know exactly what you are doing, it is safe to aspirate an effusion almost anywhere at all. So with pericardial effusion. I don't think it is necessary to do it early; the pericardial sac has almost limitless powers of distention, but if you find at any time at all that your patient is getting more cyanosed, I think it is your plain duty to aspirate. The aspiration is done as in the pleural effusion and just as safely. There are two points for aspiration, one in the fifth interspace to the right of the sternum. This is safe because the heart does not come over that far. The other is in the sixth interspace about the nipple line. This I think is quite as safe and is perhaps, a little easier. You make sure not to feel or see any apex-beat in that area and then introduce the trocar. If you get it in contact with the heart muscle it will wabble as the heart beats. Then you withdraw it a little. I don't think there is any danger to the heart. The heart muscles are well protected and there is very little danger of hemorrhage. There may be a few drops of blood, but this will be easily absorbed. Some other conditions in the pericardium bring up the possibility of hemorrhage; this is ordinarily traumatic and if very serious might be operable. There are two

forms of hemorrhage in the pericardium which are deadly; one is a rupture of the heart and the other is a rupture of an aneurysm of the aorta—one of the commonest causes of sudden death. Here is an effusion which kills; it occurs so very suddenly that the pericardium does not have a chance to dilate. Sometimes instead of getting serum in the pericardium you get pus; here you will have a leukocytosis and an up-and-down temperature, and here I operate as I do in empyema with equal chances of recovery. Sometimes in addition to the pus there is air in the pericardium and that brings about a strange condition of affairs. The pericardium is tympanitic with a curious churning sound which is due to the friction of the air. It occurs in young people with a tuberculous process at work, sometimes with involvement and sometimes limited only to the pericardium.

In adherent pericardium we are not always able to elicit a history of pericarditis. Some of the physical signs which will help in making the diagnosis are: Large area of cardiac dulness; the apex-beat displaced to the left, and the inability of the lung to cover the heart during inspiration. In many cases there is more or less recession of the apex-beat. By that I mean that the whole pericardium seems to recede. Another is where you get in the back a recession of the interspaces or Broadbent's sign which is of value, the recession of the interspace being along about the ninth, tenth, and eleventh ribs. This condition is important in diagnosis not from the standpoint of treatment but from that of prognosis. The ordinary hypertrophied heart you may put at rest or on digitalis with good results, but where you have an adherent pericarditis there is nothing you can do to help.

You probably are all familiar with the things which I have pointed out and I simply wanted to go over them and clarify them. It will remind you that there is such a thing as pericarditis. We are liable to run in grooves of thought and not infrequently we say afterward that if we had only given things more thought we would have made the correct diagnosis. These seminars are largely for this purpose to jog our minds so that we will all be on our toes and won't overlook things.

DIAGNOSIS OF AORTIC ANEURYSM

JOSEPH SAILER

IN 1844 John Erickson published his observations on aneurysm. From this it appears that Galen described a traumatic aneurysm. Actius in the sixth century also described a traumatic aneurysm. Feinelius, in 1542, described aneurysms occurring in the internal arteries, especially the chest. This was fifty years after the discovery of America, and when syphilis had been introduced into Europe. Paré, in 1582, described the aneurysmal bruit.

An aneurysm is a dilated artery. It is of no advantage to discuss the morbid anatomy nor the classification, excepting as it relates to etiology. According to the latter, aneurysms can be divided into traumatic—which may or may not involve communication with a vein—and syphilitic.

It is my intention today to limit discussion to syphilitic aneurysms of the aorta. These may occur in any part and be of any size—sometimes filling one side of the chest or half the abdomen. They may produce signs and symptoms of the greatest variety, from nothing to almost everything. There are certain important or unusual signs that deserve mention. Inspection reveals perhaps a pulsation or a tumor that projects above the surface, sometimes huge; or there may be an enlargement of an arm, or a distortion of the chest, or an edema of one side of the chest. One patient was admitted to the hospital with a vigorous pulsation of the left thorax that was synchronous with the heart. There was also a loud systolic murmur heard over the left chest with each expansile pulsation. The chest was flat, the heart displaced to the right, and an exploratory puncture with a small needle withdrew only blood. Death occurring on the third day, the autopsy disclosed a saccular aneurysm of the transverse arch

that had adhered to the left thoracic parietal pleura in the axillary region and that had ruptured. It is, I believe, the only case reported of expansile pulsation of the wall of the thorax due to this cause.

Changes in the pulse are common. Often there is a Corrigan pulse, but this is due, not to the aneurysm, but to the frequently associated aortic insufficiency. If the innominate or a subclavian artery is involved, inequalities of the radial pulses are noted, that on the affected side being smaller. The most remarkable change is the disappearance of the pulse. I reported a case of this some years ago, and suggested the term pulsus deletus. This has not been adopted, but no other designation has been suggested. Loss of the pulse was first noted by François Franck, working with Marie in Paris, but is mentioned as a clinical phenomenon by Broadbent and others. It must be understood that the pulse disappears, but the circulation is maintained; the pulse waves, presumably, being absorbed by the aneurysmal sack. The parts involved which are, of course, the limbs, are warm, they flush and blanch, bleed if cut, and have normal sensation. My experience has been that it occurs only if the aneurysm is large, and in both the cases in which I have observed it death occurred in a short time.

A pulsating tumor is not necessarily an aneurysm. It may be an empyema necessitatis—a comparatively rare tumor nowadays, although I have seen two—because empyemas are usually recognized early and promptly drained, or a pulsating sarcoma. I have seen one such tumor in the stump of the left arm, and the pulsation was more violent than I have ever observed in an aneurysm. Adherent pericardium may cause a localized pulsation in the lower interspaces in the lower left axillary line, Broadbent's sign, but this could hardly be mistaken for an aneurysm. It is said that a visible pulsation of the spleen is sometimes observed in aortic regurgitation. I have felt such a pulsation twice, but I have never been able to see it, and in both cases I was not convinced that it was an expansile pulsation of the spleen, and not an impulse transmitted from the heart. It is perhaps interesting to record that one of the pulsating

empyemas of necessity started as an abscess of the spleen. A manifestation of aneurysm that is sometimes confusing is the communication of the sack with the descending vena cava. This causes a distortion or swelling of the upper part of the body, including the arms, whilst the lower part looks shrunken. The upper part is cyanosed. The symptoms are severe. This is peculiarly a Philadelphia condition, for one of the earliest cases was observed and reported by the late Dr. William Pepper; another by Dr. Howard Fussell. Dr. Allen J. Smith performed an autopsy upon one of my ward patients, a negro woman, in which the communication had taken place without evidence of venous congestion of the head and arms, although the opening easily admitted the finger, a case for which neither of us could ever find a satisfactory explanation, unless the pressure in the aneurysm was so slight that it did not overcome the venous pressure. At Vichy I saw a patient with all the signs of this communication; that is, marked swelling from the axillæ upward, with cyanosis and shrunken trunk and legs, but whose roentgenogram failed to reveal an aneurysm, and who recovered completely under antiluetic treatment. I had thought that it was a condition that could not be simulated by anything else.

The diagnosis of aneurysm is usually easy; that is, it is a condition that comes, as a rule, to the hospital wards, and not to our offices, and, as is usual in such cases, the disease is well advanced when first seen. As I have already indicated, mistakes may be made even when there is a large pulsating tumor, but they are exceedingly rare, and the *x*-ray study furnishes valuable assistance. The important diagnosis is of the early stage, and the essential part of this is to have the suspicion aroused that an aneurysm may be present. The early signs in the order of their importance, as I estimate them, are as follows:

First, Sibson's sign.[1] This is a diastolic shock felt at the aortic cartilage. It is not pathognomonic, although it is more frequent in aneurysm than it is in hypertension; nor is it invariably present, but when felt the suspicion should arise that

[1] This name is given by Broadbent, but the sign is not described in Sibson's works.

an aneurysm may exist. It is most easily perceived if the palm of the hand is placed firmly over the thorax to the right of the sternum; and it is more distinct during expiration. It corresponds to the accentuated second aortic sound which is almost invariably present, and, as is the case with all accentuated second aortic sounds, should be transmitted or also heard in the vessels of the neck. There is, however, an important exception to this. If a loud aortic second sound is not transmitted to the carotid arteries, it means that something intervenes to prevent the transmission, and this is almost invariably an aneurysm of the ascending portion of the arch of the aorta. I have observed 2 cases in which the suspicion was aroused solely by this sign. Both were young men who had been infected in boyhood, and in both the suspected diagnosis was confirmed by other signs and *x*-ray studies. Both were small, early aneurysms that might otherwise have been overlooked. I think that it may be stated aphoristically that the failure of transmission of the second aortic sound upward suggests an aneurysm of the ascending part of the arch of the aorta.

Oliver's sign, or, to call it by a better name, tracheal tug, probably is the most valuable clue. It is not common, but requires careful technic to elicit it. The two forefingers should be placed on either side and beneath the cricoid cartilage, and this cartilage lifted by a vertical movement without pushing against it; the higher the lift, the greater the tug; if the tug does not increase, it is not Oliver's sign. I have compared it with the feeling of the fishing line when a small fish takes the bait—the kind of fish that a boy catches—first a nibble, and then a distinct pull. Tracheal tug also occurs sometimes in cases of mediastinal tumor, and indeed in any morbid process that causes adhesions in the superior mediastinum. Aneurysm should always be suspected first.

The aneurysmal bruit was first mentioned by Paré; afterward by nearly all writers. It is not constant, is usually rather late in appearance, and may be altered by manipulation. The thrill must have been noted long ago in the arteriovenous aneurysms, but apparently was not mentioned until the beginning of the

nineteenth century. Neither is as common as one is led to expect by the earlier writers.

A dull systolic thud that gives a sense of power is very suggestive of the existence and location of an aneurysm, and almost invariably is accompanied by a systolic impulse. The difference between these is usually easily recognized; the thud or shock feels as if a blow were struck inside the chest, not against the chest wall, and the impulse lifts the palpating hand. There is also often a loud systolic note heard over the aneurysm, analogous to, if not the same as, Traube's pistol-shot sound that is heard over the arteries in aortic regurgitation.

The additional signs are those of tumor in the chest; that is, pressure upon the veins, causing swelling and edema; upon the trachea or bronchi, causing stridulous breathing; and upon the ribs, producing bulging.

Displacement of the organs is common. The more easily recognized is lateral displacement of the trachea—usually a very early sign, that, like many others, occurs in other conditions and only serves to arouse suspicion. Gerald Webb once taught me an easy method of appreciating the lesser degrees of this. Facing the patient, the tip of the right thumb is placed lightly against the inner edge of the right sternocleidomastoid muscle and very gentle pressure made against the trachea. The same process is used with the left thumb. The side toward which the trachea is deflected will feel slightly firmer. The suprasternal pulse is usually present. It is merely an indication of distention, elongation, and upward displacement of the aorta, and is also common in arteriosclerosis, aortic regurgitation, and mediastinal tumor.

It may seem a mistake to describe the symptoms of aneurysm after the signs, but in my own experience, gathered chiefly in the hospital wards—for I cannot recall more than a half-dozen cases in private practice—signs have been by far the more important. There is, however, one symptom that is fairly common, often occurs early, and is always suggestive. This is pain in the chest boring through to the back, and worse at night. The unfortunate possessor of an aneurysm will be called upon to endure much

and various pain; that caused by pressure upon the nerves, the erosion of the bones, and the discomforts of dyspnea and dysphagia.

The causes of death are not many. If no other condition intervenes, the patient with aneurysm dies either of pressure, of exhaustion, or—and this is the most frequent termination—rupture of the sac. Pressure usually acts upon the trachea, causing death by slow dyspnea. It is perhaps as agonizing to watch as any fatal condition that occurs, and must be vastly more agonizing to endure. To see some miserable wretch leaning forward, well braced, dragging with great effort a little air through his reluctant trachea, and then expelling it with an equal effort, perhaps eight or ten times per minute, is certainly tragic; and there is no help, for morphin is almost immediately fatal. One of the most remarkable cases that I have ever seen died suddenly, and at the autopsy a huge saccular aneurysm had compressed the right auricle, and forced it into the tricuspid orifice, so that the circulation was suddenly stopped. For some time that heart must have been working under a tremendous handicap; and we did not recognize it, for the aneurysm seemed big enough to account for everything. The real aneurysmal death is rupture. That is the end to be expected by the physician, and the anticipation of which should be sedulously guarded from the patient. It may occur at rest or in action, in bed or abroad. The blood may be retained within the body, or spatter all surrounding objects until it seems that more than $\frac{1}{13}$ of the patient's weight must have escaped. But no matter how it occurs the end is death, perhaps delayed for a few hours or days, but inevitable.

Before I describe the treatment of aneurysms, or at least my idea of what this treatment should be, I shall try to tell what one may reasonably hope to accomplish. These hopes are three. The first is to check the progress of syphilis, the underlying cause. May I say parenthetically that I have always felt that the existence of syphilis is a disgrace to modern preventive medicine. A parasite limited to a single species for a host, that in a few years ceases, even in that host, to be actively contagious,

should be eliminated by ten years of concerted work. I shall not discuss the treatment of syphilis, but no matter how successful it cannot restore the artery to its normal caliber. We are not even sure that the destruction of all the spirochætæ, were it even possible, would prevent further dilatation. The second is to keep the patient as comfortable as possible. Iodids sometimes diminish the pain, and sometimes I have rendered the patient more comfortable by draining edema from the legs, or aspirating serous cavities, but the one dependable resource is morphin, always to be used in the later stages unless there is tracheal obstruction. The third is to delay the rupture of the sac. For this there is only one method that I know. The dry diet (Tufnell's), the injection of gelatin, the administration of calcium, the ligation of branch arteries—remember I am discussing only the syphilitic aortic aneurysms—have all been tossed into the overflowing waste basket among the discarded. The Moore-Corradi method of wiring aneurysms I believe still to be useful. This does not cure the aneurysm; it does not promote the formation of an organized clot—for the clot of an aneurysm never organizes—it does not prevent rupture, but in a certain number of cases it seems to delay it; and in many cases it diminishes for a time the pulsation, and usually reduces pain so much that the patients beg to have it repeated as soon as conditions become bad again, often after several years. Properly performed, the operation is practically without danger. Dr. H. A. Hare of this city has performed it more than thirty times without an accident; nor have I, and my reckoning is now above a score, had any complication. On the other hand, unless the proper instruments are used, unless the technic is perfect, and unless there is a skilful electrician to assist death may easily be caused. It will only be possible to give a brief description of the operation.

The wire is an alloy of gold and platinum. Gold is too soft and kinks easily; silver is too brittle and may break, while the alloy is strong, does not kink nor break easily, and coils readily. It has the disadvantage of being expensive. The needle must be insulated. This was formerly done by enameling, but it

scaled so easily that it was most unsatisfactory. With the aid of Mr. Pilling a needle was devised with a hard-rubber sheath, that has been satisfactory and durable. With strict asepsis and with the aid of a local anesthetic the needle is inserted into the aneurysmal sac, and the wire passed through as rapidly as possible until about 30 feet have been introduced. It is then at-

Fig. 200.—*A*, Hard-rubber insulating cover, permanently fixed to *C*; *B*, cone of non-ferrous metal, attached to steel tube *C*; *C*, concave-pointed steel tube attached to *B*; *D*, thin 14 K. gold wire about 30 feet long.

tached to the positive pole of a galvanic battery. A large electric pad has previously been laid under the patient's back in contact with the skin, and to this the other pole is attached. A galvanic current of 5 milliamperes is allowed to flow through the aneurysm for five minutes, then increased to 10 milliamperes, and at five-minute intervals the current is increased 5 amperes at a time until the patient experiences a slight burning sensation. The current is then stopped, the wire cut close to the needle and thrust into the cavity beyond the point of the needle by a well-fitting wire stilet, the needle withdrawn, and the patient returned to the ward.

In more than one-half of the cases the immediate result of the wiring is abolition or marked reduction in the pain. During the ensuing twenty-four hours the pulsation may rapidly decrease, until, as in one patient whose aneurysm seemed so nearly ready for rupture that the needle was inserted with considerable trepidation, the bulging was no longer in evidence, and the pulsation could not be detected. If there has been dyspnea, it is relieved; and in one case dysphagia was also cured. The general condition of the patient is also improved. One of my patients, elsewhere reported, apparently at the time of wiring, in the late stages of decompensation, orthopneic, and unable to walk, was able to return to his business. Not all cases do well. One, a negro woman, showed no improvement at any time, but was at

least no worse. She died a few weeks after the operation, and was the only case of complete failure. Occasionally the improvement is remarkable. One patient was discovered six months after wiring, pushing a huckster's cart about the Delaware Avenue region of Philadelphia, probably the worst job that he could have undertaken; but he seemed cheerful, comfortable, and, for a huckster, reasonably prosperous.

The duration of life after wiring varies. The case already reported lived three and a half years and then died as a result of unwarranted exertion. Patients with aneurysms of the peripheral arteries live longer. One patient with reasonably large subclavian aneurysm was wired a second time at an interval of seven years. He may still be alive. The most interesting of all the operations was one performed upon an abdominal aneurysm. With the assistance of Dr. T. T. Thomas, the abdomen was opened, the aneurysm reached easily, the needle inserted, and the wire introduced. There was no difficulty in the operation and the wound healed promptly. The results were only moderately satisfactory. Six months later he was wired a second time, and a short time later died of rupture. As the abdominal aneurysm when first seen was huge, it was our opinion that life was prolonged.

On the whole, no other treatment of aneurysm yields such satisfactory results.

ANGINA PECTORIS

JAMES M. ANDERS

IT has been observed that about one out of every twenty physicians dies of angina pectoris. This is not strictly speaking a disease, but rather a symptom complex. It may depend upon pathologic lesions of the heart or it may not. It may be, therefore, of cardiac origin or it may be due to lesions outside the heart (aorta), as is well known; and, indeed, it may occur in patients who present no macroscopic lesions, either in the heart or in the aorta. As a rule, we find that it attacks people with more or less hypertrophy of the heart, with narrowing of the coronary arteries, or with pericarditis (rarely), or aortic regurgitation, or finally with syphilitic aortitis. These are the lesions usually met with at postmortem in cases of angina pectoris, and two or more of them may be seen in the same case. Let us not forget that angina pectoris may occur without any macroscopic lesions whatever.

Dr. Cabot, of Boston, has made studies of the necropsies and clinical records at the Massachusetts General Hospital and has reported the results in his work on Facts on the Heart. His results are of great interest and importance. He studied 127 cases that showed narrowing of the coronary arteries; he also found 11 cases which showed no macroscopic lesions of the coronaries, and only 5 of the latter, lesions of the aorta. Of these 138 cases, the total number studied, 94 showed narrowing of the coronaries without angina pectoris; 33 showed narrowing of the orifice of one or more branches with angina pectoris, and 11 showed no narrowing of the coronary arteries, which were perfectly smooth throughout, but angina pectoris was present. I think this is most interesting. Recollect that 94 cases were found to present gross lesions, quite as gross as those that showed

angina pectoris. Some showed occlusion of the coronary branches yet no angina pectoris, and all died of other causes. They occurred in older men rather than in older women, as is true of angina in general, and nearly half of them showed a mild grade of fibrous myocarditis. Right here there is observed a difference between the cases of coronary narrowing which showed angina pectoris and those which did not. Those which did not show angina seemed to have a better coronary circulation. At all events, the myocardium was on the whole better nourished than in those which were attended by angina pectoris.

Of the 33 cases that had angina pectoris more than one-half of them had coronary lesions, some with total occlusion of one or more branches, and 9 or 10 at least out of the 33 showed syphilitic aortitis. Some of the latter showed blocking of the coronary branches also, but the majority simply aortic lesions. More than two-thirds occurred in men over fifty years of age. He noted that cases which showed angina pectoris rarely lost their attacks of angina after secondary dilatation came on following venous congestion. There is a wide-spread impression that once we get secondary dilatation the attacks are likely to subside or become less intense. Dr. Cabot's observations proved the contrary, however, because whereas 3 or 4 lost their anginal attacks, more than four times as many continued to have the attacks despite venous congestion and secondary dilatation of the heart. So we must get away from the notion that secondary dilatation is likely to be followed by disappearance of the attacks of angina pectoris. The blood-pressure showed no rule in these cases. It was increased or normal in, perhaps, about an equal number of cases. From my own personal experience I can state safely that the majority show a moderate degree of elevation of blood-pressure. The cases Cabot studied which showed no macroscopic lesions showed venous congestion at autopsy, and yet they all died of angina pectoris.

If what I have said regarding the underlying pathologic condition or conditions of this syndrome be true, we have an explanation for the fact well known to many of us who have practiced medicine for a comparatively long time, viz., that this

disease is likely to occur in persons who are subjected to severe mental and emotional strain; it is especially common, therefore, among physicians, clergymen, actors, and captains of industry. Those of you who have practiced in hospitals considerably, as well as in private, have doubtless observed the greater frequency of occurrence of this affliction in private patients, as compared with the hospital ward patients.

Among potent predisposing causes are age and sex. There is a marked preponderance in the male sex, more than two to one. From my own private practice and from the records of the Medico-Chirurgical Hospital, including case records of my associate, Dr. Jameson, I collected a total of 103 cases. Of these, 75 were furnished by my own case records, 7 by Dr. Jameson's, and 21 were from the records of the Medico-Chirurgical Hospital. I will present a division of the cases as per age-periods of five years:

15 to 20	3	46 to 50	16
21 to 25	4	51 to 55	15
26 to 30	4	56 to 60	15
31 to 35	9	61 to 65	10
36 to 40	5	66 to 70	7
41 to 45	10	70 and over	5

The average age was forty-nine years and six months. Under forty there were 25 cases, and over forty there were 78. In my series there were 69 males and 34 females.

Among other predisposing causes are the toxins; these are more important than is generally appreciated. Of my own cases there were 4 that gave a history of an excessive use of tobacco. Then there are the toxins of infections, *e. g.*, of influenza, pneumonia, and acute rheumatism. In my series there were 16 cases in which rheumatism and 4 in which influenza had preceded the attacks of angina. There were 6 cases of aortic regurgitation, and 4 of aortic regurgitation associated with rheumatism.

Pneumonia has not been mentioned as a predisposing factor by most writers on this subject in the past. Again, for years I have observed the association of rheumatism with angina pec-

toris, but never before had I gone to the trouble of making an investigation into the question of the percentage of cases in which rheumatism is associated. Undue nervous and emotional strain, before mentioned, are predisposing causes, and they are important from the standpoints of prognosis and treatment. Muscular effort is an exciting factor of first importance; in some patients even the slightest muscular effort may bring on an attack. Walking up a hill, or against a strong wind, is especially liable to bring on a paroxysm in those predisposed. Mere exposure to cold has given rise to the disease in a few of my cases; and then again, there is a considerable percentage in which gastric disturbance precedes the attack. After the paroxysm we often note eructations of gas and sometimes vomiting, but when digestive disturbance occurs before the attack, it may be noted as one of the exciting causes.

Do we know the immediate cause of the pain? No, we are still in doubt, although the most popular theory is the one suggested by Allan Burns, which is to the effect that a temporary or transient ischemia or lack of blood supply to the heart muscles is the cause of the pain. In other words, the needs of the heart muscles are not met by the collateral circulation.

Another theory, and one which to my mind is quite plausible, is that of Lauder Brunton (and others), who thought that overstretching of the ventricles was the cause of the pain in angina pectoris. The pain is similar in origin to what we see in cases of over-distention of the intestine and bladder and other hollow viscera. Allbut believed that 90 per cent. of all cases of angina pectoris were due to syphilitic and other forms of aortitis, his theory being that under muscular effort and excitement the tension in the fibrous, outer coat of the aorta becomes increased and causes the pain.

A fourth theory is that advanced by the man who originally described angina pectoris and gave it its name, to wit, Heberden; he thought that the pain of angina pectoris was caused by a cramp of the cardiac muscle. All these theories seem more or less plausible, but I think the first one mentioned is supported by the most proof in its favor.

This brings me to speak briefly of what I regard as important symptoms of angina pectoris, namely, those belonging to the prodromal stage. Not long since Kahn,[1] of New York City, published a paper in which he called attention to this subject. Of the cases I collected, the premonitory symptoms were studied. Here is the result in the 103 cases of my series: Thirty showed dyspnea on exertion; 8 precordial distress with constriction or burning on exercise; 5 palpitation of the heart; 4 sudden general weakness and faintness, and in 1 case pain in left arm. Of my series, therefore, 48 cases, or 46 per cent., presented prodromal symptoms. In Kahn's series of 82 cases, 25 or 30 per cent. presented such symptoms.

From the nature of these prodromes, it is clear that heart failure of incipient grade precedes the onset of true angina pectoris in many cases at least. This fact is of signal interest, in view of Dr. Cabot's observation to the effect that ensuing cardiac dilatation with venous congestion was not followed by either a diminution in the number of attacks or their severity in the majority of the cases.

These few more or less mild prodromal symptoms are, I think, very important from the standpoint of treatment; because if recognized, and if their gravity is well understood and the cases presenting them properly handled, they may never develop into true angina pectoris, as they otherwise would. I believe this because in a number of my own patients who complained of marked burning on exertion, I put them to bed for four weeks and when they got up the pain had disappeared on walking on the level. One of them subsequently developed pain in the left arm, but she never had true angina pectoris. I am aware that many patients do have this pain in the left arm for a long time before angina pectoris appears. I feel the preliminary symptoms should be regarded as belonging to the clinical picture of the complex, and therefore important from the standpoints of both prognosis and treatment.

I will now pass on to the symptomatology of the attack. There is intense pain, usually about the midsternal and left

[1] **Amer. Jour.** of the Medical Sciences, September 1926, p. 418.

parasternal regions, and this pain is accompanied by a vise-like feeling of constriction which holds the chest motionless; the pain radiates to the left shoulder, down along the left arm, inner aspect, to the fingers in some cases, and is often accompanied by a numbness in the arms and hands and precordium with, in the severe cases, an intense dread of death. The pain, however, instead of radiating down the left arm only, may radiate down both arms, and again it may extend into the right and not at all into the left arm. Sometimes it goes through to the back and at times to the back of the neck. I recall one case in which the most intense pain was felt in the back of the neck, coming forward and affecting the lower jaw.

These are instances of what Mackenzie has described as referred sensations. We have irritation in the heart or aorta transmitted to the visceral segment of the spinal cord whence impulses arise, which flow out along the upper thoracic nerves to the arm where they are felt as pain. When this pain extends to both arms, we have this explanation to give: viz., that the irritation may be so intense as to be transmitted from one side of the spinal cord to the other and then projected along the course of the thoracic nerves to both upper extremities and there recognized as severe pain. In those cases in which the right arm only is the seat of this radiated pain, we explain it by assuming that the irritation is situated in the nerve endings of the right ventricle.

There are certain physical signs which I think are rather important. On inspection, immobility of the body is present during the attack of pain; the respirations often seem to be suspended until the attack is over, when the breathing becomes rapid and shallow and the patient is afraid to move even after the pain has gone. The skin is cold and clammy with perspiration, and to palpation there is little expansive motion of the chest. The pulse in typical angina pectoris remains regular, and it is not increased in frequency, as a rule.

There is more or less enlargement of the left ventricle as shown by extension of the area of cardiac dulness to the left, in many cases. We often hear murmurs which had been present

before the angina pectoris developed. In aortic stenosis we hear rough murmurs over the base of the heart with a feeble second sound; but please remember that the murmur of aortic

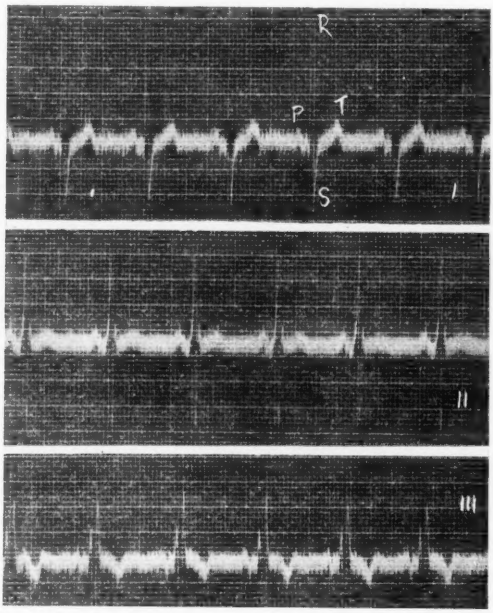

Fig. 201.—T diphasic in Lead II. T inverted in Lead III.

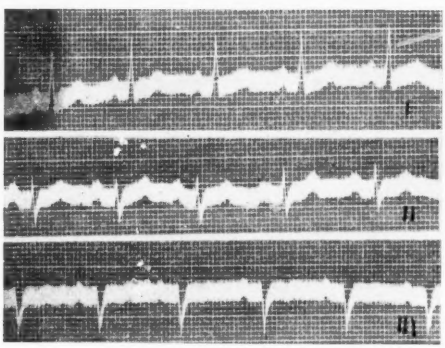

Fig. 202.—Shows left preponderance.

regurgitation is present oftener than any other in angina pectoris (vide supra). After the attack is over the physical signs may prove negative on examination. I have said that the blood-pressure observes no rule in angina pectoris. Neither has our knowledge of this affection been appreciably advanced by the use of an electrocardiograph, although inversion of the T-wave is noted in certain cases of this disease (Fig. 201). The accompanying tracing (Fig. 202) shows only left preponderance.

We have anomalous types of this affliction. For example, angina vasomotoria, which is due to spasm of the peripheral vessels; the patient feels cold and is pale during the attack; the facies may be slightly grayish in color—leaden appearance. Angina vasomotoria is not true angina pectoris, but usually develops into it sooner or later. Then we have the form known as angina abdominis, in which pain is located in the epigastrium. The only symptom which is referable to the precordium is the marked constriction or oppression. That form often develops into typical angina pectoris with precordial pain. I have seen patients suffering from this type with radiation of pain along the course of the ribs and toward the back, but not to the shoulders and down the arms.

Then there is a third subvariety—the so-called *angina sine dolore*—where there is no pain, but marked thoracic oppression, and an anxious countenance. All of these subtypes should be regarded as angina pectoris and treated accordingly.

I am going to take up briefly the question of diagnosis. The most characteristic features are the very severe retrosternal and left parasternal pain, sudden in onset with radiation to the left shoulder, down the left arm, principally its anterior aspect and the paralyzing sense of impending death. Of considerable value in diagnosis are the age of the patient, the sex, the brevity of attack, and the anxious moistened features of the patient. There are a number of cases in which one or more of the characteristic features are missing. If they are met in a man past forty years of age who has arteriosclerosis, syphilitic aortitis or aortic regurgitation, one ought to think of the possible existence of angina pectoris.

We should always attempt to distinguish between true angina pectoris and that condition known as acute coronary infarction, because in acute infarction, whether due to arteriosclerosis or thrombosis or syphilitic aortitis, we have certain symptoms which are not met in true angina pectoris. For example, there is immediate and profound shock; the systolic blood-pressure falls to 100 or lower and moist râles are heard over the bases of the lungs, and some of these cases die from edema of the lungs rather than from the cardiac condition, although death is usually attributed to the cardiac lesions. The pulse fails, is feeble and rapid. All forms of arhythmias may occur, and where one of the main branches of the coronaries is blocked death is the usual outcome, often in so-called status anginosis.

True angina must be distinguished from the functional type. Arteriosclerosis, syphilitic aortitis, or aortic regurgitation are met in true angina. The cases occur oftenest in males and at a later period of life. Per contra, the functional form may occur as early as puberty and is more common in females. Bear in mind that these functional varieties of angina pectoris often occur at night and usually without an exciting cause, and are periodic. In true angina pectoris the intervals between attacks are longer, as a rule. The pain of true angina pectoris is much more severe than in the functional form; it completely immobilizes the body. The functional form does not do this and the patient tosses about the bed. The paroxysm lasts longer in functional angina than in true angina. Please to remember further that you never see that moist, anxious facial expression in the functional form.

A number of abdominal conditions may be confused with angina pectoris, but there should be no difficulty in distinguishing them from true angina. The gastric crises of locomotor ataxia may simulate angina pectoris. Here, however, we have symptoms which do not belong to angina pectoris at all, such as lack of co-ordination, the Argyll-Robertson pupils and absent reflexes; true, we may have vomiting, but it does not occur at the end of the attack only as in true angina pectoris. On the other hand, both vomiting and eructations of gas occur during the attack in the gastric crises of locomotor ataxia.

I want to speak briefly of one condition which I have observed to be confused with true angina pectoris. I refer to cholelithiasis, which is sometimes mistaken for angina, because the pain in the epigastrium is associated with more or less precordial distress and discomfort. As a rule, in cholelithiasis the pain radiates to the back and sometimes rises as high as the right shoulder. In that affliction you see the patient tossing about, or rolling on the floor, trying in all manner of ways to find a comfortable position. This is not true of angina pectoris; the patient does not move about, but is immobile for the time being. The attack of pain is longer in cholelithiasis, it may be followed by jaundice; in any suspicious cases we ought to try to complete the diagnosis by finding biliary calculi in the stools or by getting a picture of the gall-stones with the *x*-ray.

There are cases of thoracic aneurysm which bear a clinical resemblance to angina pectoris. Those of you who have had much clinical experience are aware that many symptoms of thoracic aneurysm referable to the chest in that disease are not met in angina; for example, the brassy cough, the inequality of the pupils, and differences in the radial pulses. Do not fail to have an *x*-ray examination made. I never saw a case yet in which, if carefully done, the *x*-ray did not show a pulsating tumor in the course of the aorta in thoracic aneurysm.

The prognosis is distinctly unfavorable as a general rule. I have long felt that it helps a great deal in attempting to formulate a prognosis to remember the different courses pursued by this disease. Four leading subvarieties were described by Osler: (*a*) "The fulminant or rapid form with one or two attacks only, or sometimes with the condition known as status anginosus; (*b*) the form with a duration of one and a half to two years and a varying number of attacks; (*c*) the chronic form, lasting upward of ten years with a varying number and growing intensity of attacks, that, for instance, of John Hunter, whose first seizure occurred twenty years before his death; and (*d*) lastly, the small group of cases which may last for months, or as long as two years, with attacks of great severity, and yet may completely recover."

I suggest that to these four groups we add a fifth: (*e*) those

cases presenting prodromal symptoms (dyspnea and palpitation, burning and precordial distress, and feeling of constriction on exercise). These cases, if properly treated, may never develop into true angina.

The treatment has two main objectives; the first is to get the patient over the attacks as safely and comfortably as possible. This we do with the use of either amyl nitrite (strength m.v) or nitroglycerin tablets. The physician should always instruct the patient in the use of these two agents. The patient must carry amyl nitrite ampules with him, and if he experiences an oncoming attack, he should immediately break an ampule on a pledget of cotton or handerchief and inhale it. The dose can be repeated if relief is not obtained from one ampule. The doctor does not often see the patient during an attack in private practice. Nitroglycerin may act better than the amyl nitrite. I have found a hypodermic tablet freshly prepared (gr. 1/100) placed under the tongue, a satisfactory mode of administration. The dose may be repeated every half-hour, if required to relieve the paroxysm. If the nitrites fail to relieve the patient, then I resort to the hypodermic use of morphin and atropin, gr. $\frac{1}{3}$ to $\frac{1}{2}$ of the former and gr. 1/150 of the latter, repeated after an interval of a few minutes if not relieved. Most attacks are over in from a few seconds to a couple of minutes. During the intervals between the attacks, the main objective to be reached is the prevention of another attack by avoidance of the exciting factors, and the removal of all predisposing causes. We know the exciting causes, and in cases where a little exertion even causes an attack it is better not to allow the patient to go around, but put him at absolute rest.

I have seen cures which have lasted for years effected by rest in the recumbent posture for from one to two months. If the predisposing cause be syphilis, treat it with arsphenamin in minute doses at first, and then increase them until you are employing the average dose used in that affliction. The only exception to this rule is in cases complicated with advanced nephritis. Mercury is not to be omitted from the treatment of these cases.

Gout or rheumatism, when they are present, are to be treated actively and those cases in which digestive disturbances seem to bear a causative relation to the attacks require very careful attention both as to diet and medication. We should also look for foci of infection, and if present remove them whenever possible. The iodids are most valuable in the treatment of the accompanying arteriosclerosis. We said formerly that the iodid of potassium was a useful remedy when syphilis was the cause, but I have found it a useful remedy in many cases where the Wassermann reaction was negative. I have seen it produce a quite remarkable effect upon the frequency and severity of the seizures a number of times in recent years. My custom has been to employ small doses, except in those cases in which lues can be shown to play the chief etiologic rôle, when the dose should be gradually increased from 2 or 3 up to 15 or even 20 grains *ter in diem*. Nitroglycerin is of value in the arteriosclerotic type, given over longer or shorter periods of time; it tends to soften the arterial walls. Nitrite of soda also seems to have a good effect in quickly recurring attacks.

In concluding, if I have tried to impress you with anything in the nature of new or useful information, it has been to call attention to the importance of the recognition of the prodromal features, as part of the symptom complex, and of treating the patient presenting them before the case develops into true angina pectoris; also to stress the fact that angina pectoris is always indicative of myocardial failure, even though the lesions accounting for this condition are not invariably macroscopic.

THE CIRCULATION IN RELATION TO SURGERY

THOMAS McCRAE

ANY discussion of the consideration to be given to the state of the circulation in relation to surgery and obstetrics must concern itself with a problem which has many sides. The question may be considered in various ways and the subjects for discussion may be chosen under different heads, but this matters little if we touch on the most essential points. I take it that we are mainly concerned to bring the patient safely through an operation or delivery, and in this the factor of the circulation is important. We may consider (1) the state of the patient beforehand with especial reference to the circulation; (2) the operation itself in which the anesthesia is particularly important, and (3) the subsequent circulatory conditions which may arise. The surgeon's part of the problem is an important one, as it bears on the duration of the operation, the amount of blood lost and the extent of trauma sustained; but these points hardly concern this discussion. There need be little mention of emergency operations which may have to be done despite serious circulatory conditions. Here the best course cannot be chosen, but the lesser of two evils selected. Evidently every measure which can be adopted to aid the circulation is of particular value in these cases.

1. **The Condition of the Patient.**—It is evident that in this aspect of the question a thorough examination of the patient is important. We must all have been amused to see an anesthetist listen with his ear or stethoscope to a patient's heart just before beginning the anesthetic. This may have some value from its influence on the patient who may think that it is a precaution for his welfare. Some gross change may be so discovered, but as a means of knowing the state of the patient's circulation it

has little or no value. If there is any marked departure from normal which could be found by this procedure it should have been made out beforehand by a proper examination. It should not require emphasis that a study to determine the condition of the circulation is just as important as one made to exclude pulmonary tuberculosis before giving ether.

It may aid if we try to separate certain groups, realizing that all patients cannot be put within definite boundaries. There are certain problems which come up fairly often, among which is the patient with a *frank valvular lesion*. To some of us this group seems important, but in reality, with one exception, it is not so. A valvular lesion has little bearing in itself in adding to the risk of anesthesia and operation, unless it is aortic insufficiency. With this lesion there is undoubtedly more risk, but in the patient with good compensation it is not easy to estimate it. Some might say that aortic stenosis should be regarded as adding to the risk, but it is difficult to decide as this is such a rare lesion. If there is good compensation, it does not seem that it should be regarded as particularly serious. Mitral valve lesions with good compensation can be regarded as adding little to the risk. Some might hold that mitral stenosis demands special consideration, but if the compensation is good and there has not been any previous decompensation the additional risk is slight.

The Patient with Frank Myocardial Insufficiency.—Here we are on very different ground and we must regard such patients as distinctly poor risks. It does not seem possible to lay down any rules for the members of this group, as each patient is an individual problem. Certainly every effort should be made to study them thoroughly, and in this the use of the electrocardiograph should take an important place. It may show evidence of disturbances—for example, branch-bundle block—which could not be recognized otherwise.

One may well ask: What about the patient with unrecognized myocardial disease? We are all aware of the difficulty of deciding as to the absence of myocardial disease, but if a proper study is made the number of cases in which doubt remains

should not be very large. But a proper study means a careful history, with special attention to possible infections and careful observation of the patient. An electrocardiographic study should be made if possible, as it may show evidence of disease otherwise not to be diagnosed. Certain groups of patients are suggested at once as demanding special care, such as the obese, those with arteriosclerosis, those who have had thoracic pain, even if we feel reasonably sure that it is due to neurotic disturbance, and those with anemia. But with every care it is well to realize that there are some patients who have myocardial degeneration which we cannot recognize readily. Still we should have our suspicions aroused and so be stimulated to the greatest possible solution of the problem.

The most important point is to determine the condition of the myocardium and in this to ascertain how the heart carries its load, especially any extra load, is essential. There is much help to be obtained from the history if the patient is reasonably intelligent and the physician knows how to obtain the information. One might say that even an unintelligent patient can give useful information if he is properly questioned. The *response to effort* is the important point, and most patients have been carrying on tests of this in their daily lives. We can try them by various tests, but I submit that the tests of ordinary life are usually just as valuable if we use them.

Elderly Patients.—In the majority of this class we have to deal with arteriosclerosis, some degree of myocardial degeneration, and kidneys with different grades of efficiency. There is one side of the problem in this class which we often forget and that is the fact that they have reached advanced life, which means that they had a good circulation to begin with. So far as it goes we have good material—although old—with which to work. In one group—males with an enlarged prostate—there is in addition, very frequently, urinary tract infection and fairly marked renal disturbance. The operations fall into two groups, one in which it is emergency matter, as for strangulated hernia, and the other in which there is some time for preparation, as in the case of enlarged prostate. When in Baltimore I had the opportunity

of seeing with Dr. Hugh Young many of his patients who belonged to the latter group. It was very remarkable to see how the risks of operation could be reduced by previous care. The special indications can be grouped as follows: (1) To bring the circulation into the best possible condition. (2) To improve the renal function and bladder drainage. (3) To secure proper rest and sufficient sleep for the patient in the few days before operation. This last is very important as many of these patients are wakened frequently by the desire to urinate and may have gone for long periods with little rest.

The improvement of the circulation demands study of the conditions present. In general it may be said that a period of rest, with careful diet and proper purgation—but not drastic purgation—and usually digitalis in moderate doses seem to meet the indications.

Cardiac Irregularity.—It is natural that in patients with some form of arhythmia the question should be raised of the safety of anesthesia and operation. It is possible to diagnose the nature of these with fair certainty and to assess the risk intelligently. As to *sinus irregularity* and *extrasystoles* it may be said that, if other findings are not contraindications, they may be disregarded. *Auricular fibrillation* and *auricular flutter* must be viewed with grave concern. In general, one would regard patients with these conditions as being serious risks and in them only an emergency operation, necessary to save life, should be done. The same may be said of *heart-block* in all the forms, including branch-bundle block which can be diagnosed only with certainty by the electrocardiograph. *Pulsus alternans* should be regarded as representing serious disease. Some hold that this applies particularly when it occurs with a pulse-rate which is not much increased and that it has much less serious significance when the heart-rate is rapid. In the subject of *paroxysmal tachycardia* the other conditions are the important ones. If there is evidence of myocardial disease, caution should be exercised. With a good myocardium the risk is probably very slight. Cardiac irregularities, which are to be regarded with apprehension, are those due to serious disease of the myocardium.

Arteriosclerosis.—No general statement can be made as to how much this increases the risk of operation by itself. The condition is so variable in its anatomic distribution that the common conclusion drawn from palpation of the radial artery is practically worthless. As many arteries as possible should be examined, especially the femoral and the arteries at the ankles. The condition of the aorta should be studied. It may be palpable in the episternal notch or in the abdomen, or enlargement of the thoracic aorta may be demonstrable. The retinal arteries are important also. A man may have arteriosclerotic peripheral arteries and show very little change in the visceral vessels, or *vice versa*. The former alone has no importance in this connection.

What is really of importance is the condition of the myocardium and kidneys. If either or both of them show evidence of disease, the problem is the wider one and not the narrow one of arteriosclerosis alone.

Blood-pressure.—Hypertension must be studied in its relation to the state of the circulation as a whole and the condition of the kidneys. If hypertension exists with a sound circulation and good kidney function it adds little to the risk, although we can never be sure of the condition of the cerebral arteries except as the retinal arteries are an indication. There may be a possibility of the rupture of a cerebral vessel or a small aneurysm during the anesthesia. The loss of blood during the operation is often a great safeguard. *Hypotension* seems important only if it is due to myocardial weakness. In itself it does not seem to be of any great moment.

Vascular Disease Due to Syphilis.—This deserves special mention as there is no question that it adds greatly to the danger of operation. There is the strong chance of disease of the aorta and possibly of the coronary arteries. This in turn means myocardial degeneration. A careful study should be made of such patients before they are passed for operation.

Special Tests.—The question arises as to whether we can determine the matter of operative risk by any special tests. In the use of these there are advantages and disadvantages. The

use of any test which suggests that the circulation is not efficient and calls attention to possible danger is an advantage. If it is employed as part of a thorough examination and properly considered it is useful. But if it is thought that one test or more taken alone is sufficient without a proper study of the patient it may be of disadvantage. A number of these tests have been devised, some simple, others more complicated.

Moots determines the "pressure ratio" by dividing the pulse pressure by the diastolic pressure, which gives a normal figure varying between 40 and 60. If the pressure ratio is high or low there is some danger. With figures between 25 and 75 there is probable safety, if outside 25 and 75 there is danger. No such rule can be regarded as of invariable application. The factors which are responsible for high or low figures should be evident from the general study of the patient. But if such a test directs attention to possible danger it is useful.

The simple test of determining the time during which the breath can be held is of value and has the great virtue of simplicity, as has the Moots test. After remaining quiet for five minutes the patient takes an average full breath, but not as deep a breath as is possible, holds the nostrils, and keeps the mouth closed. The time during which he can do this is recorded. The average normal is from 40 to 50 seconds. This test may draw attention to possible danger.

As to rules which may draw attention to danger arising during operation, McKesson has stated certain points which are of value. In a patient whose circulatory records have been normal at the beginning of the operation, a pulse-rate of 120 or over, a pulse pressure of 20 or less and a diastolic pressure of 80 or less, suggest that shock has occurred, and if this condition persists for half an hour there is great danger. For estimating the degree of circulatory disturbance he regards as safe a rise of 10 to 15 per cent. in the pulse-rate with no change in the pressure, or 10 to 15 per cent. decrease in the pulse-pressure without any change in the pulse-rate. There is danger if there is 15 to 25 per cent. increase in pulse-rate and 15 to 25 per cent. decrease in blood-pressure. The condition is probably fatal with an increasing

pulse-rate above 100 with a falling blood-pressure of 80 systolic or below, and 20 or less pulse-pressure for more than twenty minutes.

2. **Anesthesia.**—We cannot enter into controversial subjects here, but the question of the circulation in surgery cannot be properly answered unless we give due weight to this. An anesthetic acting on the central nervous system interferes with the operation of the various stimuli which are concerned in the regulation of physiologic processes. These influences may be dangerous. First let us discuss the influences on respiration, as this is closely associated with the circulation, and disturbance in one function at once influences the other. We are familiar with the frequent occurrence of cyanosis during anesthesia, or, in other words, defective oxygenation. We used to regard this as due to the respiratory center being less sensitive to the stimulus of an increase in the hydrogen-ion concentration of the blood; but is this correct? We know that periodic breathing is a sign of oxygen lack. Haldane, Meakins, and Priestly[1] suggested that the oxygen percentage in different alveoli varies considerably. The alveoli with a higher percentage than the average are not able to saturate the hemoglobin to a higher point with oxygen than blood alveoli with an average oxygen percentage. But the alveoli with a lower oxygen percentage than normal are unable to saturate the blood to anything like the normal amount. Therefore, when the depth of respiration is diminished or the oxygen in the inspired air is decreased, there is oxygen lack, although the mixed alveolar air may have a normal percentage.

That such conditions are frequently present in the ordinary anesthesia is not to be doubted. An oxygen lack is present on many occasions and from this we know that results follow rapidly. A more extreme degree of oxygen lack is responsible for the symptoms in carbon monoxid poisoning. In this the gravity of the condition depends partly on the degree of oxygen lack, but also on its duration. If the latter is prolonged, permanent damage of cells results. Minor grades of oxygen lack cause headache, depression, nausea, etc., and these may persist long after the anox-

[1] Jour. of Physiol., 1919, 52, 433.

emia is over. Another point to be noted is that the symptoms of oxygen lack may not appear until after the period of such lack is over. This should be kept in mind when postoperative conditions arise of which the explanation is obscure. In all grades of anoxemia the symptoms may persist long after the period of oxygen lack is past. We attribute these symptoms to damage done to cells by the deficient supply of oxygen, and especially to the cells of the central nervous system. The circulation through the central nervous system in man at rest is rapid, and also large in comparison with other parts of the body. The tissues have no reserve of oxygen to draw upon and any lack in the circulating blood can cause damage in a short time.

Two other conditions may operate, namely, acidosis or alkalosis. In deep anesthesia, owing to the respiratory center being less sensitive to its normal stimulus, the carbon dioxid pressure in the arterial blood and the hydrogen-ion concentration increase. With this there is a decrease in the alkali reserve of the blood, but this is not evidence of increased carbon dioxid pressure in the blood. An increased carbon dioxid pressure is not necessarily harmful. A decreased alkali reserve in deep anesthesia may be due to the respiration being unduly stimulated by oxygen lack, harmful in itself, and resulting in a delay in the anesthetic being removed from the body after the anesthesia is over. What is the bearing of these points on the problem of the circulation? It is difficult to be dogmatic, but the possibilities of damage seem to be evident. The dangerous possibilities are handled safely by the body mechanism in the vast majority of cases, but many must come close to the line of safety. It seems that we have not appreciated the importance of the dangers of anoxemia sufficiently in the past. Certainly every anesthetist should realize them.

The Effect of Anoxemia on the Heart.—The studies of Resnik[1] show that the auricular ventricular conduction time is altered. This depends on several factors, the degree of anoxemia, the length of time it operates, the heart-rate, and the general condition. Interventricular conduction is little interfered with in

[1] Jour. Clin. Investig., 1925, 2, 93, 117, and 125.

normal hearts. In a heart already the seat of branch-bundle block it is reasonable to suppose that the effects may be more marked. The sino-auricular node is highly sensitive to anoxemia. In the auricles, "relatively the refractory period is decreased and conduction slowed, in the early stage of anoxemia, so that a circus movement may develop; in the later stages of anoxemia the reverse is true." It seems reasonable to conclude that in hearts already the seat of disease, the possibilities for acute disturbances of various kinds are always present if anoxemia occurs.

3. The surgeon's side of the problem needs only a reference. The length of time taken for the operation is important, especially with reference to the anesthesia. Yet there are surgeons who consider that the time element is of slight importance if there is little blood lost. There can be no question of the importance of the amount of hemorrhage and the extent of trauma to the tissues. It is not necessary to point out how these influence the circulation and so the operative risk.

It is evident that the problem of the circulatory risk in an operation must vary, owing to a number of conditions. In an emergency operation there is no time to improve matters and the best possible must be done under the circumstances. In these cases it is especially important that the anesthetic be given with the greatest skill and particular care given to the prevention of anoxemia if a general anesthetic is used. When time permits a proper study of the problem should be made. If there is evidence of circulatory disturbance, an effort should be made to estimate the risk and to lessen it so far as this is possible.

In the cases in which matters go wrong with a patient who is apparently healthy, one is suspicious that in some cases the health was only apparent, and that a careful examination with a proper history would have shown that the condition was not normal. But there are undoubtedly some cases in which no method of examination can give warning of danger.

The Treatment of Postoperative Conditions.—It is evident that no set rules can be laid down for this as the picture differs. One point is very definite and that is that there is too much

haphazard treatment. There should be a careful study of the problem presented. Particular mention must be made of the improper use of digitalis in patients who show disturbance of the circulation after operation. Because the heart is rapid, the first sound feeble and the blood-pressure low, it is often thought that digitalis should be given. Too often it is given in large doses by injection when there is not the least indication for its use. To speak generally the great indication for digitalis is myocardial insufficiency, and before we give it we should be sure that this exists.

The patient should be carefully studied and if possible the cause of the disturbance determined. In many cases the difficulty is in the peripheral circulation, vasomotor paralysis, and in such cases to give digitalis is to make matters worse. What the myocardium lacks most is more pressure to work against, and you do not provide this by giving digitalis. If loss of blood is largely responsible, transfusion of blood is probably of the greatest aid. As to measures which increase the peripheral resistance we are only too well aware of how few we have which are of value. Epinephrin is of some value, but ephedrin promises to be of greater use. It should certainly be given a thorough trial in such cases. As to the aid to be expected from strychnin it is difficult to speak. Certainly it has lost its vogue to a large extent. Bandaging of the extremities should be of value, and it may be that we have got too far away from the use of this simple measure. In the same line would come tight bandaging of the abdomen, if this has not been done already by the dressings. This may help to lessen the collection of blood in the splanchnic system.

Digitalis is indicated if there has been myocardial insufficiency previously, but in this case the condition should have been recognized and sufficient of the drug given beforehand. It is perfectly good practice to continue its administration.

Obstetrics.—The problem most frequently encountered is to decide whether a pregnant woman with cardiac disease should be allowed to go to full term or whether the pregnancy should be terminated. No rules can be laid down, as each problem is

an individual one. There are three groups: (1) The patient's condition of myocardial insufficiency is so marked that there is no question of the necessity of interference. (2) The patient has such good compensation that there is no indication to terminate the pregnancy. In a certain number of this group the patient is anxious to avoid the addition to the family and hopes that a cardiac lesion may seem a sufficient reason for terminating the pregnancy. (3) The patients in whom it is a nice problem as to the best course to adopt. It is with them that any discussion must deal particularly.

In the last group a careful study of the whole problem is essential. Importance should be given to the results of observation, with proper treatment, for a few days. In the presence of symptoms of myocardial insufficiency, the results of treatment should be prompt and striking if the myocardium has reasonable reserve. In this event it is usually safer to wait, but if little or no improvement results, the future course of the pregnancy is not likely to improve matters and its termination is advisable. The results of an electrocardiographic study are sometimes of value and this should be done wherever possible. In general it seems to me that our attitude should be to demand proof that the pregnancy cannot go on, or, in other words, that we should be against intervention until its necessity is demonstrated beyond doubt.

If a woman near the end of pregnancy shows signs of loss of compensation, it is usually wiser to try to improve the condition of the circulation rather than to bring on immediate delivery. The result of a short period of proper care is the best indication as to the proper course. It is evident that in patients with a damaged myocardium, very careful watch should be kept on their progress with careful supervision and proper therapy.

Summary.—There is nothing mysterious about the determination of the risk in operation due to circulatory disease. It demands the same care in examination as should be invariable in all studies of the circulation. There is no rule by which we can determine absolutely the risk of anesthesia and operation. Our effort should be to determine the condition of the circula-

tion as accurately as possible, exactly as we would in any other condition. But it should be realized that what is particularly important and also additional is to determine how well the heart is going to stand an extra load, for that is what an operation represents. It is not always in our power to determine how heavy the extra load is going to be, and it is safer to estimate on the basis that it will be heavier than the ordinary expectation. It is the duty of the physician to put the heart in the best possible position to carry the extra load and in this a few days' rest and preparation are all important. It is the duty of the anesthetist and surgeon to make the load as light as possible. The question of the maximum safety or the maximum risk in a patient with circulatory disease is not to be answered by one of the three—all must co-operate.

THE VALUE OF AN *x*-RAY EXAMINATION IN THE INTERPRETATION OF HEART LESIONS[1]

EUGENE P. PENDERGRASS

THE fluoroscopic and radiographic examination of the heart is one of the best methods used in the study of heart conditions, when combined with a thorough physical examination. Interpretations of the *x*-ray findings should be made only after correlation of the radiographic and clinical data.

Fluoroscopy is of the utmost value, and the following data can be determined at such an examination:

1. Variation in size and shape during systole and diastole, the heart being studied in all directions, including the anteroposterior, oblique, and lateral directions.

2. The variations of the positions of the heart during the excursions of the diaphragms, especially during forced inspiration and expiration.

3. Pulsation—normal, abnormal, and transmitted.

4. Orthodiagraphic measurements of the heart, made by the central ray method.

The radiographic examination is important, because it gives a permanent record of the appearances of the heart and allows one to see more clearly slight variations in the outline that might be overlooked at the time of the fluoroscopic examination.

TECHNIC OF *x*-RAY EXAMINATION

1. The target film distance is 2 meters. This reduces the distortions of the heart shadow to a minimum.

2. The patient should be examined in a standing position with the front of the chest against the plate changer, the shoulders thrown forward, which carries the scapulæ out of the lung fields.

[1] From the Radiological Department of the University Hospital, Philadelphia, Pa.

The hands are placed on the hips with the thumbs backward in order to facilitate the above position. If the standing posture is not advisable for a given patient, the examination can be made in the sitting position, but never in the recumbent (Figs. 203, 204).

3. The patient should be carefully placed so that the resultant

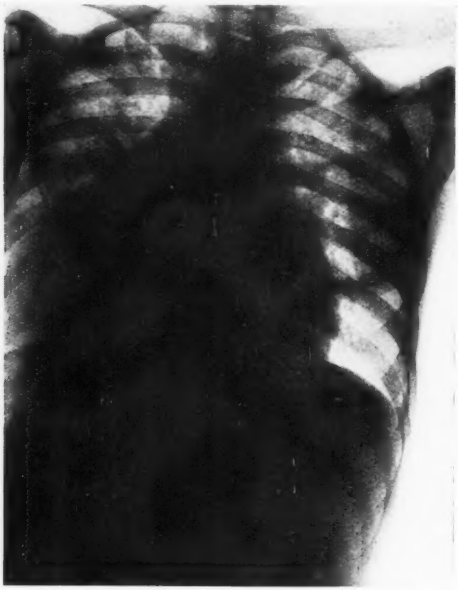

Fig. 203.—An enlarged heart with passive congestion of the lungs. This examination was made in a semirecumbent position in bed. Compare this appearance with that of the same patient made in the usual erect position. Target film distance = 30 inches.

film will demonstrate the sternal ends of the clavicle equidistant from the spine.

4. The exposure should be examined at the end of deep inspiration. It is well to have some fixed technic about this particular phase of the examination, so that if films are made later for comparison the same factors can be used.

5. The chest should also be examined by the lateral view,

with the left side next to the plate changer. This allows one to obtain the anteroposterior diameter of the heart and information about the aorta.

6. Quick exposures are essential, and the factors should be adjusted so that a thick patient can be examined in one-tenth second exposures and thin individuals in one-twentieth second.

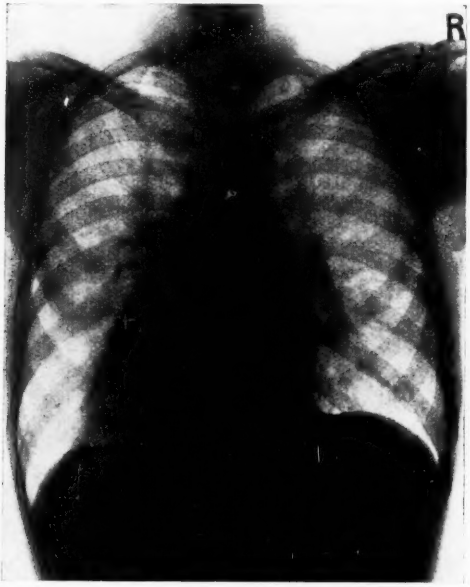

Fig. 204.—Same patient as Fig. 203. This examination is made in the erect posture, and the size of the heart is considerably less, the diaphragm lower, and the entire prognosis and interpretation are different. Target film distance = 2 meters. The above illustrations show very well the differences in appearance described in the text and a possible discrepancy between clinical and *x*-ray findings.

This permits one to obtain a film of the heart at rest and eliminates the usual blurring of the heart outline. With the heart at rest, fairly accurate measurements of it can be made. Of course, it is not technically possible to tell whether the heart is taken in systole or diastole in general laboratory work, but this is possible in the experimental laboratory.

7. The clinician should make his examination in the same posture that the roentgenologist makes his. This will allow a proper correlation of data, especially as to size.

8. The tube should be centered over the eighth thoracic vertebra so that distortion shall be reduced to the minimum, thus allowing one to make comparative deductions from subse-

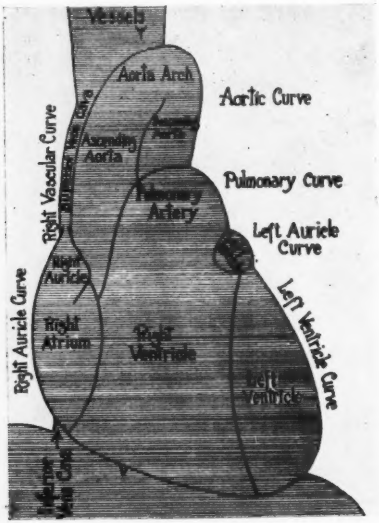

Fig. 205.—The diagram showing the different portions of the heart borders as seen in the *x*-ray film.

quent examinations. This is possible if one follows an established technic.

The Normal Heart.—The silhouette of the heart in the anteroposterior direction presents two borders which are made up of the following curves (Fig. 205):

The left border:

(*a*) The pulmonary curve is made up of a composite shadow of the pulmonary artery and veins.

(*b*) The left auricular curve is made up of the left auricle and atrium.

(*c*) The left ventricular curve consists of a small portion of the left ventricle, most of which is located posteriorly and is seen in the lateral view.

The right border:

(*a*) The right auricular curve which constitutes most of the border of this side is a composite of the auricle and atrium.

The lateral view of the heart demonstrates two borders: the anterior and the posterior (Fig. 206).

Fig. 206.—Lateral view of a normal heart. The retrocardiac space is readily seen as is the preaortic space. The small area under the horizontal arch is distinctly visible with the hilum shadow overlying it. The heart occupies the anterior two-fifths of the diaphragm and the anterior one-half of the chest. Note the straight line extending downward indicated by the arrow, which is assumed to be a composite shadow of the pericardium reflected to left diaphragm, inferior vena cava, and the posterior phrenopericardial ligaments. Distortions of the appearance of this shadow is of diagnostic significance in precardial effusion and adhesive pericarditis.

The anterior border is made up mostly of the right ventricle below and a small portion of the right auricle above.

The posterior border is made up mostly of the left ventricle below and the left auricle above.

In interpreting the heart shadow and any deformities thereof, it is well to designate them by their corresponding curves, *i. e.*, the heart shadow is enlarged with increased preponderance of the left ventricular curve.

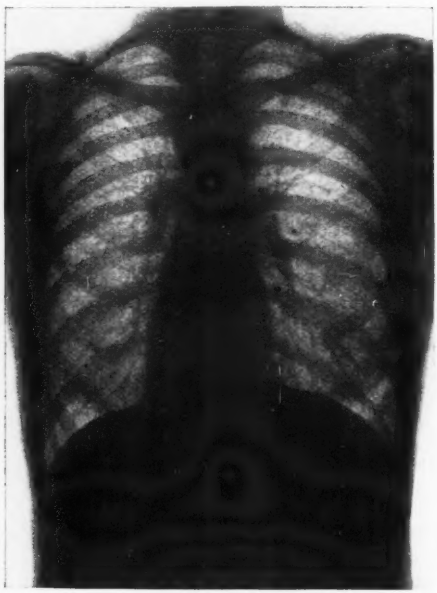

Fig. 207.—This illustration demonstrates the type called the dropped heart. It occupies the midline of the chest. The right and left borders are almost equidistant from the midline of the spine.

Size, Type, and Location.—The size of the heart silhouette varies in different individuals, depending particularly upon the build and the height of the left dome of the diaphragm. We follow the elastic rule that any heart that is not greater than one-half of the total transverse diameter of the chest is within

the normal limits of size. Practically, however, we are confronted with three main types of hearts:

(*a*) *Dropped Heart.*—The silhouette of this heart is very small. The heart hangs in the midline and the right and left borders are almost equidistant from the midline of the spine. The transverse diameter is usually less than a third of that of the chest. This type of heart is seen in the long-chested individuals (Fig. 207).

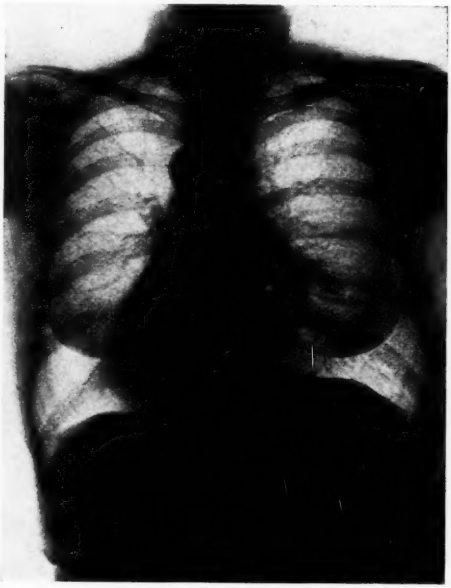

Fig. 208.—Heart in a woman aged forty-nine, showing what is regarded and described as the average heart.

(*b*) *The Average Heart.*—The silhouette of this type is slightly less than one-half the transverse diameter of the chest. It is usually seen in the young adult of average build, represented by a young man or woman of twenty years of age. A small portion of the right border is seen to the right of the border of the spine. Most of the heart shadow is to the left of the spine (Fig. 208).

(*c*) *The Sthenic Heart.*—The silhouette of this type is quite large, and is probably due entirely to the elevated left dome of the diaphragm. It is seen in stout individuals in whom the up-and-down diameter of the chest is considerably lessened because of the elevated domes of the diaphragm even after deep inspiration (see Fig. 222).

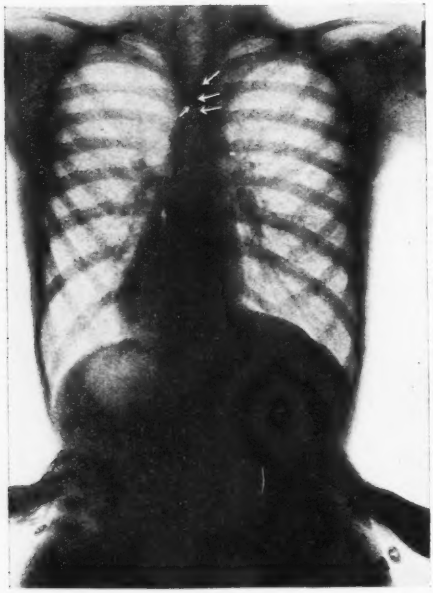

Fig. 209.—Young girl aged eleven years. Roentgen study requested because of cyanosis since birth. Thought to have congenital heart lesion. Heart outline and action normal. Increased aëration of both lungs due to congenital stenosis of trachea. This illustration demonstrates the usual midline position of the heart in children.

The apex location, as indicated in the roentgenogram, corresponds closely to the location found clinically, except that during deep inspiration it is either behind the sixth rib or sixth interspace. Laterally the apex in the dropped heart is to the right of the middle of the left dome of the diaphragm. In the average heart it is approximately at the middle of the left dome of the

diaphragm which corresponds closely to the clinical landmark, ½ inch within the midclavicular line. In the sthenic type of heart the apex is outside of the middle of the left dome of the diaphragm. The right border of the heart is slightly to the right of the spine in the anteroposterior view except in children in whom the heart occupies nearer a midline position than in adults (Figs. 209, 210).

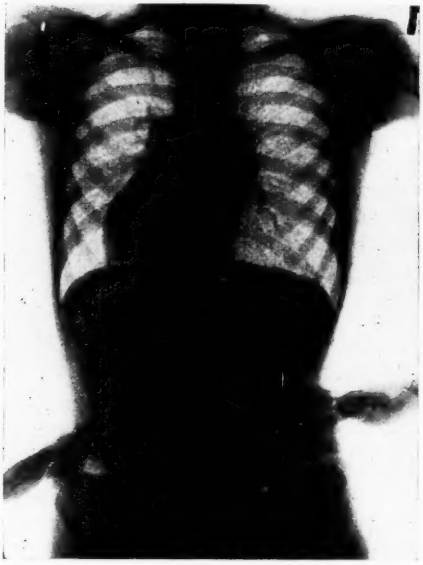

Fig. 210.—Roentgenogram of a girl aged thirteen years. This heart does not occupy as nearly the midline as seen in a number of children.

In the lateral view the heart occupies approximately two-thirds of the space between the sternum and anterior border of the spine except in emphysematous individuals (Fig. 206).

The anterior edge of the heart shadow is in contact with the anterior chest wall for a variable distance from below upward. It is somewhat less in males than in females. In the older individual and the emphysematous patient the heart is less in

contact, which is probably accounted for by the anterior bowing of the sternum and the kyphosis of the spine.

The heart shadow rests upon the composite shadow of the domes of the diaphragm, usually occupying an anterior two-fifths.

Behind the heart there is a space which is approximately rectangular in shape and a deformation or obliteration of this space has considerable diagnostic importance.

THE AORTA

Anteroposterior Position.—The aortic shadow does not extend to the right of the vertebra normally, but does extend to the left always (Fig. 208). The upper limit of the aortic shadow varies in different individuals. In stout persons it extends up to the fourth thoracic level, in long-chested patients the upper limit is as low as the sixth thoracic vertebra. In the average case it is opposite the fifth thoracic vertebra. As in the heart, the silhouette of the aorta varies somewhat with the age, size, and habitus of the individual being examined.

The aorta in the thorax consists of the ascending, horizontal, and descending arch and the descending thoracic aorta. We wish to call attention again to the fact that the term "transverse arch" is a misnomer, and this portion should be designated as the horizontal arch. This portion of the aorta extends from before backward and not from side to side as the name "transverse" implies.

Lateral View.—Such a view should always be made. Some examiners prefer the oblique view, but in our Clinic we have found the direct lateral view more valuable because there is no superimposition of dense structures in such a view, and it clearly demonstrates two spaces, the preaortic and the retrocardiac (Fig. 206). Any encroachment on these spaces may be of pathologic significance. In this view one can best demonstrate the space beneath the horizontal arch, and in good films a large portion of the descending arch and thoracic aorta are visible.

The pre-aortic space is found in front of the aorta and behind the sternum and above the cardiac shadow. This space is ir-

regularly oval, having the greatest width from before backward above, and is pointed below (Fig. 206). It is increased in emphysema by the anterior bowing of the sternum and increased aëration of the lingual tip of the left lower lobe of the lung. The

Fig. 211.—Large aneurysm of the descending thoracic aorta, destroying large portions of the last four thoracic vertebræ and the first and second lumbar vertebræ. There was no history of aneurysm in this case. The patient was examined because of pain in the back following the lifting of a heavy object. In addition to the destruction of the bodies of the vertebræ, one can see the faint outline of the large aneurysm.

space is encroached upon in dilatation of the ascending arch of the aorta.

The retrocardiac space is found behind the heart, and is irregularly rectangular in shape. An aneurysm of the descending thoracic aorta would cause encroachment upon it (Figs. 211, 212).

Fig. 212.—Same case as Fig. 211. The film was made in the anteroposterior direction and one can see the faint shadow of the aneurysm.

THE PERICARDIUM

The pericardial sac closely covers the heart and a portion of the ascending arch of the aorta. The pericardium is firmly attached to the diaphragm, and over the left dome there is a triangular reflection posteriorly that can be demonstrated best in the lateral view of the chest with the left side next to the film. (Fig. 206). The shadow that extends down from the heart to the left dome of the diaphragm and to the central attachment of the right dome has been thought to be due to pericardium, phrenopericardial ligaments, and inferior vena cava—the composite making the definite shadow that is so easily seen in a well-made film. If the above premise is correct, distortion of the normal curve should be of great value in diagnosing pericardial effusion and differentiating it from an enlarged heart. We have

found this to be true and believe it to be a classical sign of pericardial effusion.

MEASUREMENTS OF THE HEART AND AORTA

There are several methods in general use for measuring the heart. The following are used in our clinic:

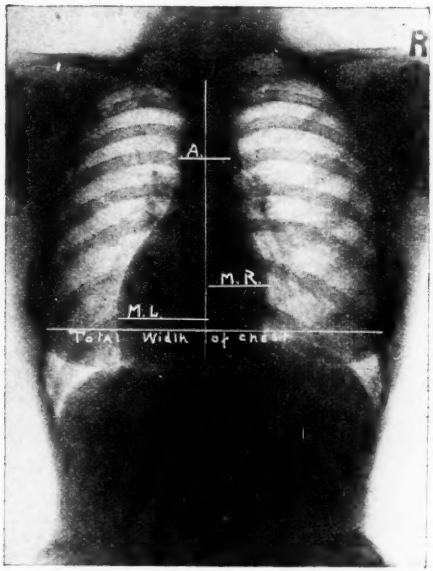

Fig. 213.—Teleroentgenogram of heart demonstrating the measurements. A, Aorta, line on level of sixth rib; *M.R.*, greatest diameter of right heart from midline; *M.L.*, greatest diameter of the left heart from the midline. The sternal ends of the clavicles are equidistant from the spine. The midpoint is found and a perpendicular line is dropped through another midpoint obtained by measuring the inside diameter of the chest just above the domes of the diaphragm. This method serves very well, except in a marked degree of kyphoscoliosis. The spine should never be used as a midline because so frequently there are minor degrees of scoliosis.

The **orthodiagraphic** method is carried out by cutting down the diaphragm opening through which the *x*-rays are emitted, thus obtaining the central ray. With a wax pencil in the right

hand and the handle of the movable *x*-ray tube in the left, the central ray is projected along the borders of the heart, and they are marked with the pencil on the glass covering the fluoroscopic screen. After the procedure has been finished, a transparent piece of paper is placed over the screen and the tracing is copied for a permanent record. There are several objections to this method:

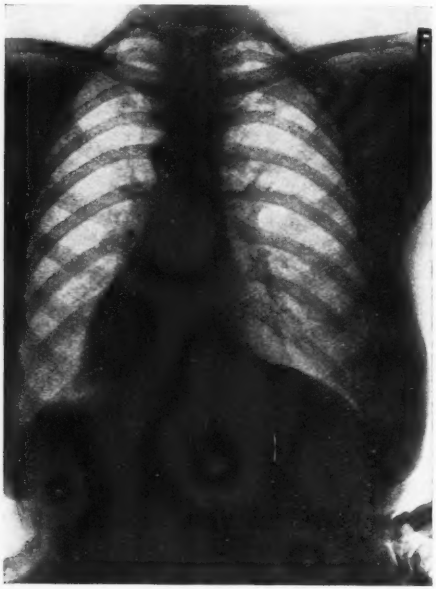

Fig. 214.—Illustration demonstrating a teleroentgenogram of the heart. There is considerable blurring of the outline in this case, thus demonstrating the difficulty in determining where one should measure. Compare with Fig. 213.

1. One must have a co-operative patient who will remain absolutely still.

2. Some patients are weak and cannot stand long enough to have this procedure carried out as it requires at least five minutes.

3. The diagram must be made without reference to the phase of respiration.

4. When there is a co-existant pleural effusion it is sometimes difficult to determine the exact border of the heart.

5. It is not easily adaptable to the sitting posture.

The **teleroentgenogram** is a film made of the heart at a tube target film distance of 2 meters, the tube being centered over the eighth thoracic vertebra. The resulting film being measured as shown in Fig. 213. The measurements are made of the aorta, heart, and total width of the chest, the inside of the ribs being taken for the landmark for the total width of the chest.

In addition we have found the lateral view of the chest at the same distance invaluable. This gives the anteroposterior diameter of the heart (Fig. 206).

In making measurements of the heart, it is necessary that the border be sharp and well-defined in order to obtain relatively accurate results. A fuzzy outline implies at once a certain amount of error (Fig. 214). It is impossible with our present apparatus to obtain a heart outline in known systole or diastole. Therefore, if subsequent examinations are made and small variations are found, one should keep in mind when rendering a report that one heart might have been taken in diastole at one time and at another in systole. This would at once allow a possible error of 1 to 2 centimeters.

CONGENITAL LESIONS OF THE HEART

The more frequent congenital lesions are:

1. Congenital pulmonary stenosis and atresia of the pulmonary artery.
2. Patent foramen ovale.
3. Imperforate ventricular septum.
4. Patent ductus arteriosus.
5. Dextrocardia.

Only the last two conditions can be recognized by the *x*-ray examination.

Patent Ductus Arteriosis.—This condition can be recognized fluoroscopically because there is an increased amount of expansile pulsation over the pulmonary area. The film made of a heart

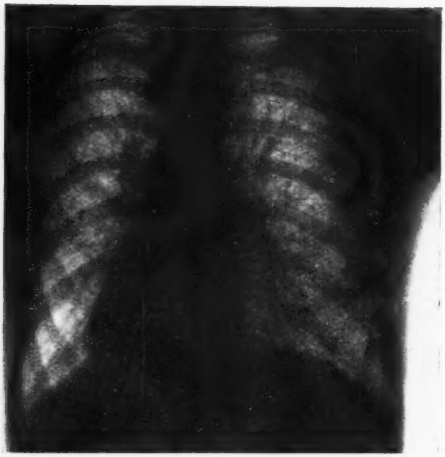

Fig. 215.—This illustration demonstrates beautifully the increased preponderance of the pulmonary curve. There was increased pulsation of this area at the fluoroscopic examination. A diagnosis of patulous ductus arteriosus was made and this agreed with the clinical diagnosis of the case.

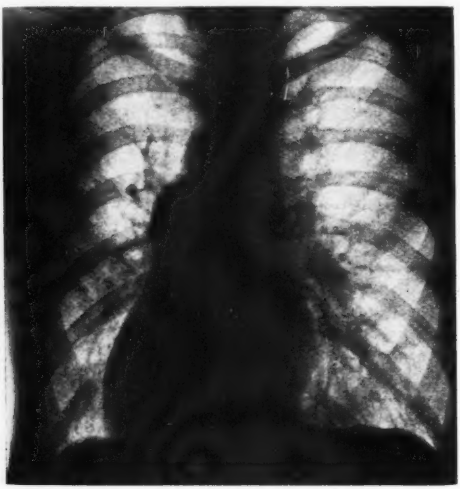

Fig. 216.—The illustration demonstrates the appearance caused by a patulous ductus arteriosus in a man aged twenty-four. The appearance might also be due to an aneurysm of the pulmonary artery.

with this lesion will demonstrate beautifully the increased preponderance of the pulmonary curve (Figs. 215, 216).

Dextrocardia will be discussed with cardiac displacements.

ACQUIRED HEART LESIONS

Enlarged Heart.—The diagnosis of an enlarged heart by the *x*-ray examination is relatively easy, but, it is very difficult to identify the cause of the enlargement (Fig. 217). This can be done only by the clinical examination, and it is only by the correlation of data that the roentgenologist has attempted in some

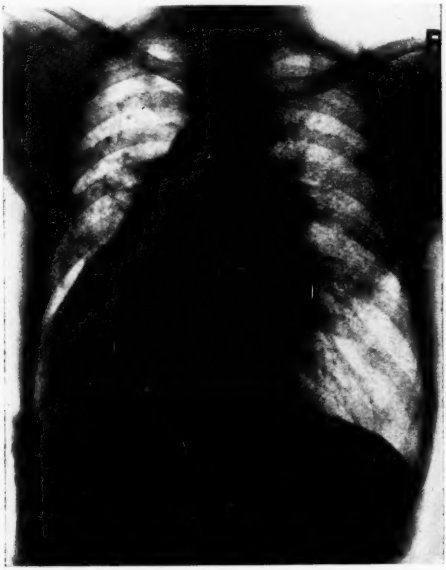

Fig. 217.—Enormous enlargement of the heart in a patient of forty-seven years of age and with a strongly positive Wassermann. There is increased preponderance of all curves. The heart is more than one-half of the transverse diameter of the chest.

instances to say that an enlarged heart of a given shape is probably due to mitral stenosis. Some enlarged hearts are enlarged only in one direction, therefore one should be competent to suggest the lesion causing the enlargement (Fig. 218). Other hearts

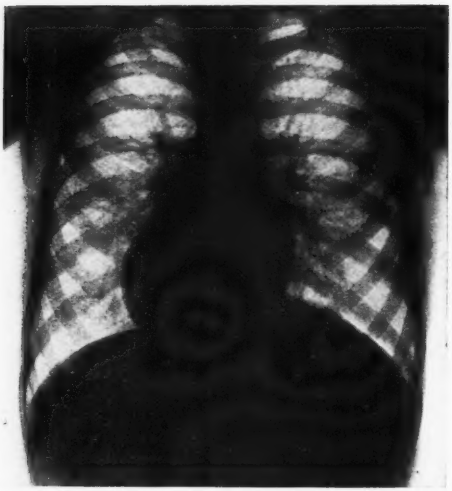

Fig. 218.—An enlarged heart in a boy of fifteen following acute rheumatic fever. All curves are increased in preponderance. Note the individual preponderance of the curves on the left side. One can distinguish the pulmonary, auricular, and left ventricular curves.

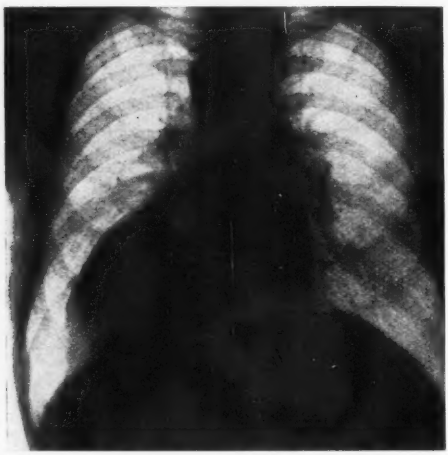

Fig. 219.—Man, age forty-eight. Patient has aortic regurgitation and the illustration shows an enlarged heart with left ventricular preponderance, which is characteristic of aortic regurgitation.

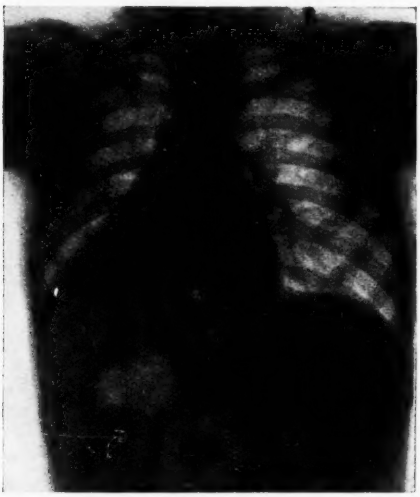

Fig. 220.—Enlarged heart in a boy of twelve who has had endocarditis over a period of four years. He has had recurrent attacks of decompensation and now has aortic regurgitation, which probably accounts for the increased preponderance of the left ventricular curve.

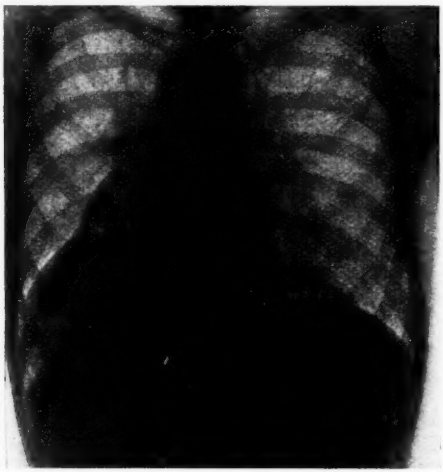

Fig. 221.—Male, age thirty-seven, with mitral stenosis and aortic regurgitation. The illustration shows beautifully the left ventricular and left auricular preponderance.

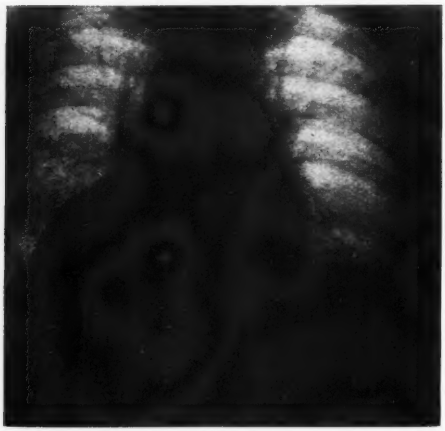

Fig. 222.—Heart of a man age fifty-six who was quite heavy, and in this roentgenogram it was thought that the increased width of the heart was due to the high diaphragm, but the lateral view demonstrates the true condition. (See Fig. 223.)

Fig. 223.—Lateral view of same patient as shown in Fig. 222. This demonstrates beautifully the increased width of the heart, which is due to an enormous left ventricular preponderance and differentiates the condition from a possible normal heart due to the high diaphragm.

are enlarged in all directions and in these cases it becomes a difficult problem to distinguish, as, for example, between cor bovinum and pericardial effusion. A discussion of the differential diagnosis will be considered under pericardial effusion (Figs. 219, 220).

Summary of Effects of Dilatation and Hypertrophy on the Heart Outline.—(*a*) *Dilated Left Ventricle.*—The heart is more

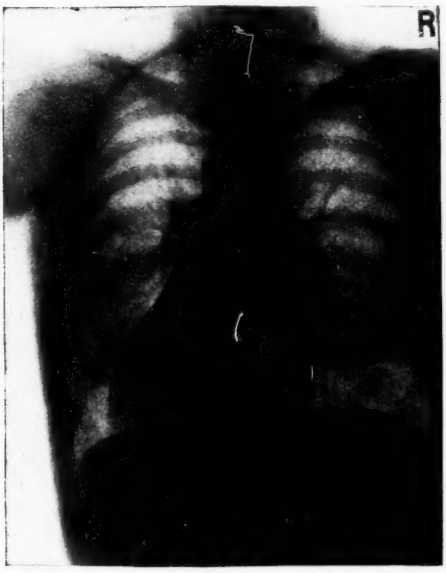

Fig. 224.—Typical case of thyroid heart. These hearts show very marked expansile pulsation and are easily recognized during fluoroscopic examinations. Almost invariably the heart action is rapid and shows no evidence of possible increased vagus action or vagotonia.

horizontal and egg shaped. The left ventricular border is more convex and the apex blunt and rounded.

(*b*) *Hypertrophied left ventricle* gives the same appearance as above, except that only the left heart is enlarged. This is seen in aortic insufficiency.

(*c*) *Mitral Stenosis* (*Early*).—Enlargement of the left auricle only (Fig. 221).

(*d*) *Mitral Insufficiency.*—Enlargement of the left auricle, and in addition the entire heart is enlarged.

(*e*) *Aortic Insufficiency.*—Left ventricle enlarged.

(*f*) *Cardiorenal Disease with Hypertension.*—Left ventricular hypertrophy (Figs. 222, 223).

Thyroid Heart.—The heart and aorta in thyroid disease may or may not be within normal limits of size, but almost invariably in hyperthyroid patients the hearts present a most unusual type of expansile pulsation. It is very noticeable, and the patients, being apprehensive, exaggerate an already stimulated heart. In this type of heart one will observe wide variations in size during systole and diastole (Fig. 224).

SECONDARY SIGNS OF CARDIAC DISEASE

Passive Congestion of the Lungs.—This is a frequent complication of decompensating hearts, and in children not infrequently is the forerunner of bronchiectasis (Figs. 225, 226).

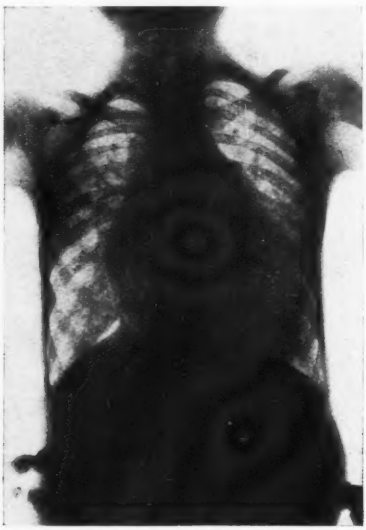

Fig. 225.—Boy, age nine, who has had a long history of cardiac disease. The heart is markedly enlarged with preponderance of all curves. There is evidence of passive congestion and some bronchiectasis.

Passive congestion is evidenced in the radiograph by increased prominence of the trunk shadows, especially those draining the lower lobes. In a severe decompensation there may be patches of drowned lung in the bases due to the edema and improper aëration of the lungs. The appearance is very similar to bronchopneumonia and cannot be differentiated except by the clinical history (Fig. 227).

Fig. 226.—Lateral view of same case as Fig. 225. This shows the enlargement of the heart in the anteroposterior direction and the prominent trunk shadows.

Passive congestion must be differentiated from bronchopneumonia, chronic bronchitis, and first and second stages of silicosis. The history is the important link in differentiation as all of these conditions give the same *x*-ray appearance.

Pleural Effusion.—Another secondary manifestation in de-

compensating hearts is pleural effusion which may be unilateral or bilateral. It is always located at the base, and extends up along the lateral chest wall and may extend into the interlobes. When a pleural effusion is present, it often obscures the heart

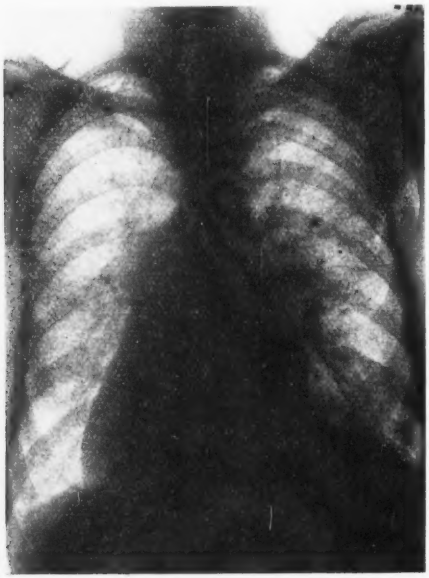

Fig. 227.—Man, age twenty-seven. Has a long-standing heart lesion. Cough and dyspnea with the slightest exertion. The roentgenogram demonstrates an enlarged heart with increased preponderance of all curves, especially the pulmonary and left auricular. In addition, there is a marked enlargement of the hilum shadows and increased prominence of the trunk shadows and linear markings, probably due to passive congestion. Conclusions: Heart lesion, probably patulous ductus arteriosus, passive congestion of the lungs with resulting hypertrophy of the heart muscle, causing the moderate enlargement of the heart.

outline and prevents one from obtaining a clear film (Figs. 228–230).

Enlarged Liver.—The last secondary manifestation of decompensation is the enlarged liver which is evidenced by an elevation of the right diaphragm. There are a number of conditions

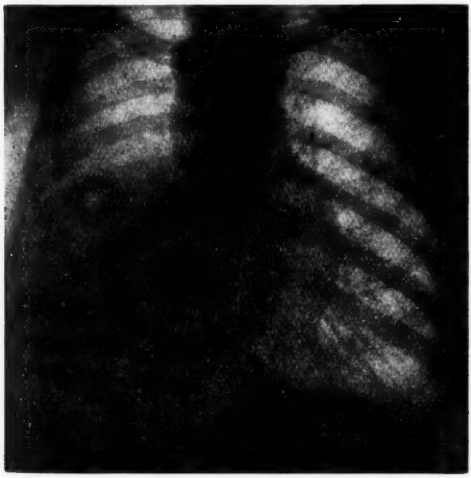

Fig. 228.—Man, age thirty-six. It was thought that the patient had a pleural effusion displacing the heart to the right. (See Fig. 229.)

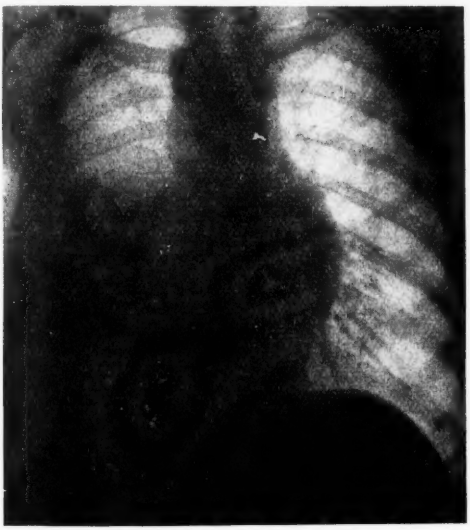

Fig. 229.—Same patient as seen in Fig. 228 after removal of 325 c.c. of fluid. Note that the heart is in the same relative position. We, therefore, concluded that the pleural collection at the left base was not entirely responsible for the increased preponderance of the right border of the heart. (See Fig. 230.)

Fig. 230.—Same case as Fig. 229. Note in the oblique view the aneurysm of the ascending arch is clearly visible. It involves the portions within and without the pericardial sac.

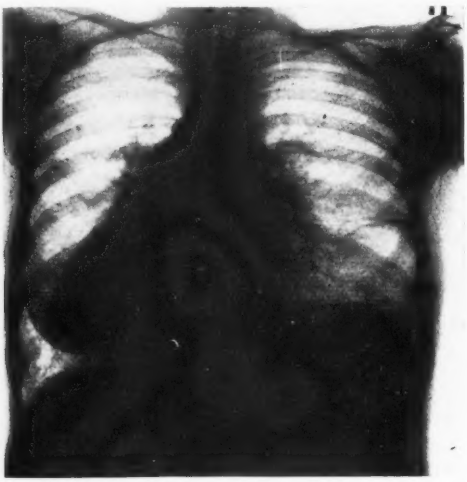

Fig. 231.—Female, age thirty-five years. The patient clinically has mitral stenosis. The film shows a rather typically shaped heart found in this condition. There is an increased preponderance of all curves, especially the left ventricular and left auricular curves. There is passive congestion of the lungs and an enlarged liver indicated by the increased height of the right dome of the diaphragm.

that elevate the right diaphragm, such as gumma or a primary tumor of the liver and a subphrenic abscess. These conditions must be differentiated from an enlarged liver due to passive

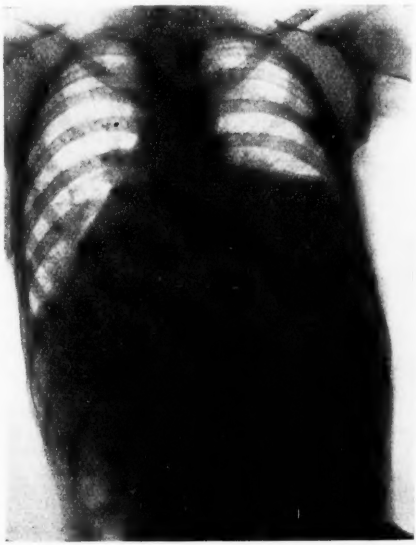

Fig. 232.—Girl, age twelve, showing an enormous enlargement of the liver in cardiac disease. The appearance at the right base looks as if it might be caused by a pleural effusion, but it is due to the enlarged liver.

congestion. The history of the present illness usually suffices to differentiate the conditions. Sometimes an enlarged liver is overlooked when there is a pleural effusion at the right base obscuring the right dome of the diaphragm (Figs. 231, 232).

PERICARDIAL EFFUSION

This is one of the most difficult diagnoses for a roentgenologist to make. There have been a number of diagnostic signs suggested as follows:

1. Enormous enlargement of the silhouette of the heart which becomes triangular in shape with the base down.

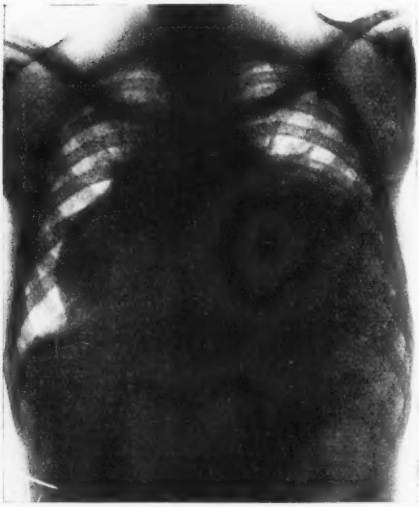

Fig. 233.—Anteroposterior view of an enormous pericardial effusion.

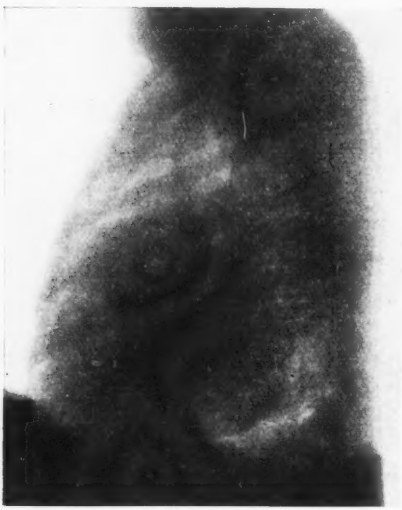

Fig. 234.—Lateral view of same case as seen in Fig. 233. Note the curve formed by the pericardial sac. The curve is convex posteriorly instead of concave, as seen in Fig. 206. It also encroaches on the retrocardiac space.

2. The acute angle formed by the heart and the right dome of the diaphragm is obliterated.

3. Another view is that the acute angle becomes more acute.

4. There are those who think the heart shadow can be differentiated from the surrounding pericardial effusion. We are of the opinion that the possible two shadows are caused by the overlying right and left bronchi.

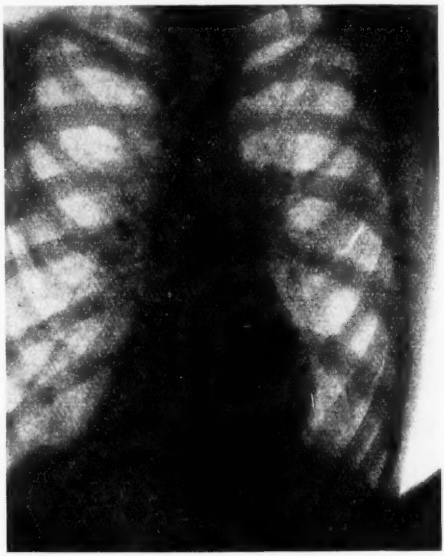

Fig. 235.—Man, age twenty-three. The illustration shows preponderance of the pulmonary curve and adhesions of the pericardium to the diaphragm and passive congestion of the lungs. At autopsy the patient had dilatation of the pulmonary artery, hypertrophy of the left auricle, and adhesions of the pericardium to the diahpragm with passive congestion of the lungs. This patient was cyanosed. Another interesting feature was a congenital deformation of the chest.

5. Another suggestion is to have the patient lie on one side or the other and the heart will float and present pulsation where it was not seen before.

We have not found any of the above suggestions entirely satisfactory for diagnosing pericardial effusion except the first,

and one is easily misled by even that. An enlarged heart with enormous dilatation can give all of the above signs.

The two signs that we have found extremely valuable are as follows:

The heart should be examined as usual with the anteroposterior and lateral films, the front of the chest and left side being next to the film, respectively. In the anteroposterior view any or all of the above-described appearances may be present. In the lateral view the normal curve of the reflection of the pericardium to the left dome of the diaphragm is concave posteriorly. In pericardial effusion, even in small collections, the reflection is convex posteriorly and encroaches on the retrocardiac space. This occurs in cases of pericardial effusion that are too small to give the other appearances (Figs. 233–235).

The second sign is only valuable when very quick exposures are made. In pericardial effusion the left border of the silhouette of the heart is distinct and the right border of the heart is fuzzy. Our premise for this appearance is increased transmission of the pulsation on the right as compared with the left, because of more effusion on the left side where the pericardium is not attached to the heart. This can be noticed fluoroscopically.

In an enlarged heart both borders are either distinct if the exposure is quick enough, or both borders are fuzzy if the heart action is too fast for the usual exposure of one-twentieth of a second.

AORTA

The lesions of the aorta that can be discovered by an *x*-ray examination are:

1. Dilatation.
2. Widening of the arch.
3. Aneurysm.
4. Calcification.

Dilatation of the ascending arch is easily detected. The aortic shadow extends to the right of the spine in the anteroposterior film, and in the lateral view the dilated portion of the ascending arch can be recognized by comparing it with the horizontal arch. Dilatation of the aorta must be differentiated from

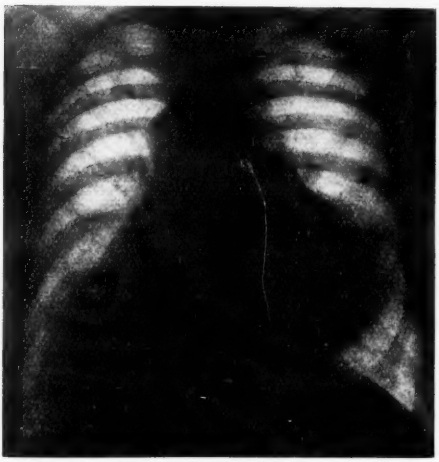

Fig. 236.—A man aged thirty-nine. Has a strongly positive Wassermann and signs of an aneurysm. This anteroposterior view demonstrates beautifully the large aneurysm arising from the ascending arch of the aorta. It was presumed that the aneurysm was filled with a large clot because there was no expansile pulsation.

Fig. 237.—Same case as shown in Fig. 236. This lateral view demonstrates the lesion directly against the anterior chest wall, but there is no bony involvement from the aneurysm. If it were thought wise to wire such an aneurysm, this view would be invaluable in giving the necessary information for such a procedure.

widening of the arch. The latter condition is usually seen in elderly people with beginning arteriosclerosis. The ascending arch in this condition may extend to the right of the spine, but the lack of dilatation is readily recognized in the lateral view.

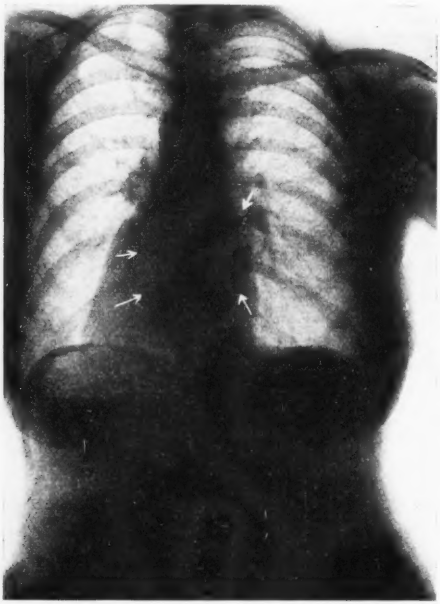

Fig. 238.—Woman aged forty-one, with aneurysm of descending thoracic aorta. Note the dense shadow outlined by arrows seen through the heart shadow.

Aneurysm of the aorta is easily detected in most instances, whether it arises from any portion of the arch or the descending thoracic aorta. The fluoroscopic examination is important in the differential diagnosis because one must differentiate between expansile and transmitted pulsation. A large saccular aneurysm filled with a clot may give little or no pulsation (Figs. 236, 237).

The aorta should always be examined in two views: The anteroposterior and the lateral. The lateral view is particularly valuable in aneurysm, especially where wiring is to be considered.

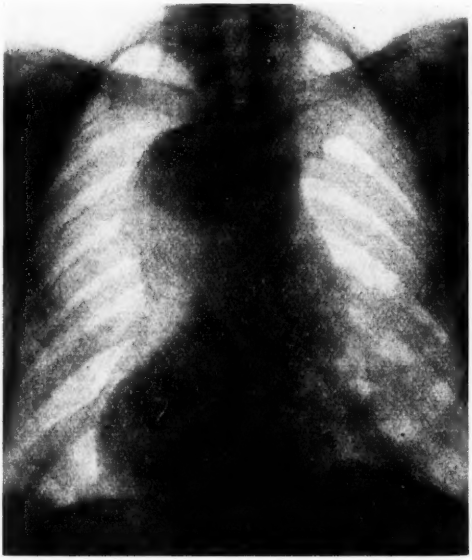

Fig. 239.—Man aged sixty-eight. Aneurysm involving the entire arch.

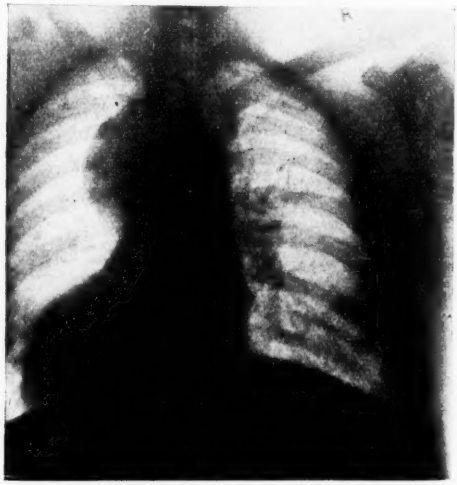

Fig. 240.—Man aged thirty-eight. Aneurysm of the horizontal and descending arch of the aorta. Note the displacement of the trachea to the right.

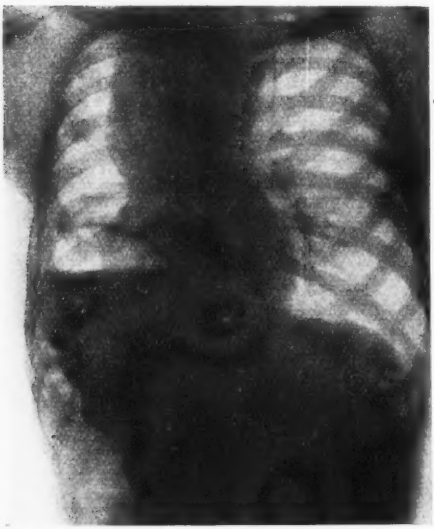

Fig. 241.—Male, age forty years. Wassermann strongly positive. The patient has a large aneurysm of the ascending arch which markedly displaces the trachea to the right. Note the patient also has an eventration of the left dome of the diaphragm. It was assumed that this was acquired from pressure of the aneurysm on the phrenic nerve.

Fig. 242.—Same case as Fig. 241. Lateral view. Note the calcified periphery of the aneurysm and how close it comes to the anterior chest wall.

It allows one to see how close the aneurysm comes to the anterior chest wall, and whether or not there has been any erosion of the bony thorax (Figs. 238–242).

Calcification of the aorta is sometimes seen and is an evidence of arteriosclerosis (Fig. 242).

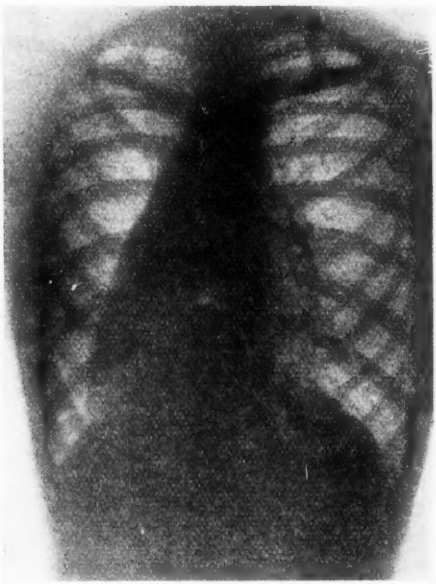

Fig. 243.—This illustration demonstrates beautifully how one should be very careful to correlate clinical and radiographic data before arriving at a diagnosis. The appearance of the heart and mediastinal shadow suggests the possibility of an aortic lesion, but the patient was a young woman of twenty-three years of age and had a temperature with a fluctuating mass over the mediastinum. The appearance was caused by a tuberculous mediastinitis.

Aneurysm of the aorta and other aortic lesions must be differentiated from mediastinitis and primary growths in the mediastinum. A very confusing lesion is a mediastinal tumor that grows beneath the arch and causes widening of the arch around it. The history and encroachment upon the space below the arch in the lateral view will greatly aid in the diagnosis (Figs.

Fig. 244.—Examination made after operation and drainage of the mediastinum. Note the surrounding lung reaction. Same case as Fig. 243.

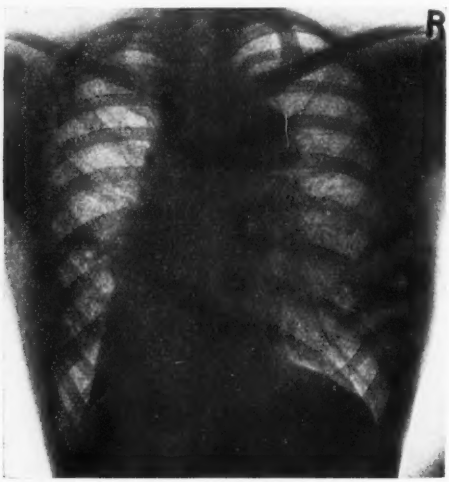

Fig. 245.—Same case as Fig. 244. Nine months after operation. Note the recurrent glandular enlargement in the region of the mediastinum.

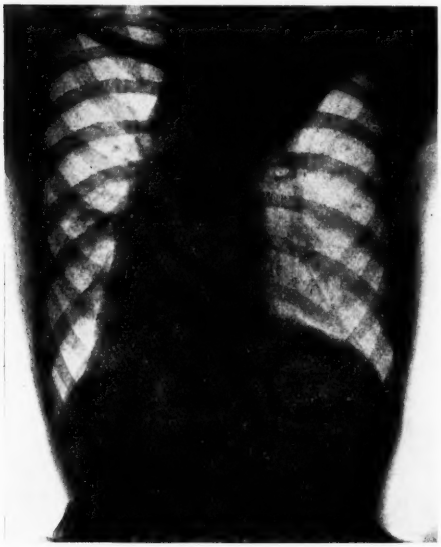

Fig. 246.—Same case as Fig. 245 two years later. There is a tuberculous pneumonia of the right upper lobe.

Fig. 247.—Same case as Fig. 246. Lateral view showing the involvement of the right upper lobe. Note the well-marked pericardial reflection of the left diaphragm.

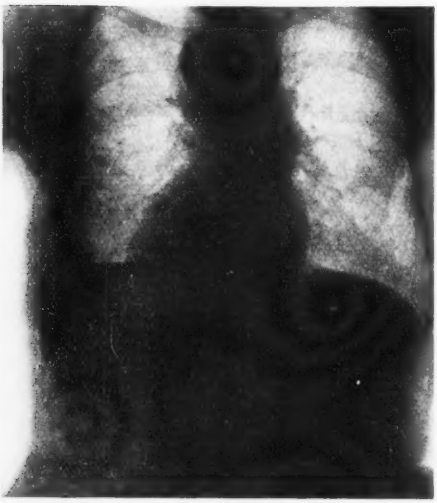

Fig. 248.—This illustration shows a dilatation of the innominate artery, calcification of the horizontal arch, and an old tuberculous process in the right apex.

Fig. 249.—Lateral view of same patient shown in Fig. 248. Note the widened arch of the aorta and tortuosity.

243–247). The same appearance in seen in mediastinitis and central empyema. The pulsation in a mediastinal tumor is transmitted unless the tumor is growing beneath the arch. Then it is expansile because the normal aorta is seen pulsating.

Aneurysm of the pulmonary and the innominate arteries do occur, but they are rare. An aneurysm of the pulmonary artery will cause preponderance of the pulmonary curve (Figs. 248, 249).

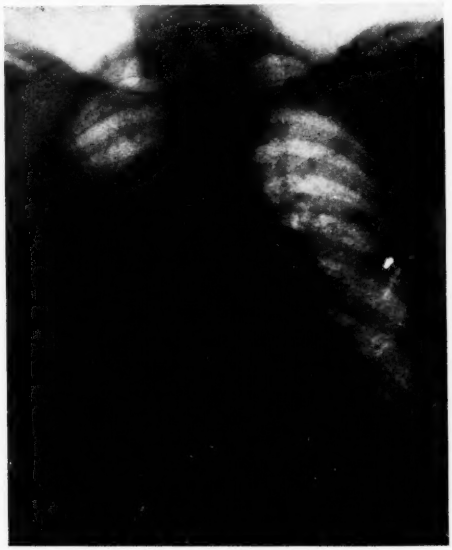

Fig. 250.—Male, age sixty-three. This illustration is included to demonstrate what was diagnosed enlarged heart, *aneurysm of the innominate* artery, and pleural effusion displacing the heart to the right. The patient died a week after this examination and the autopsy showed that the patient did have an enlarged heart with a pleural effusion. The shadow in the region of the innominate was caused by an organized clot from a dissecting aneurysm of the arch of the aorta. The clot dissected up around the innominate between the media and adventitia.

An aneurysm of the innominate artery is seen high up along the right border of the spine. This condition is easily detected, but must be differentiated from a substernal extension of the thyroid (Fig. 250).

DISPLACEMENT OF THE HEART

Displacement of the heart may be seen in the following conditions:

1. Dextrocardia { true (Figs. 251, 252). / pseudo- (Fig. 253).
2. Fibrosis following an extensive tuberculosis of one lung.
3. In organized pleural exudates (Figs. 254–256).

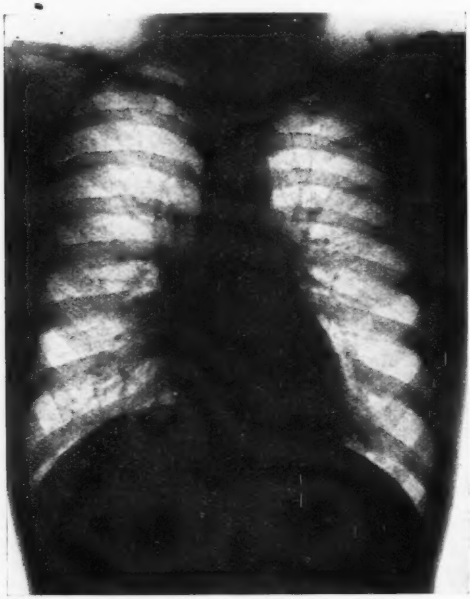

Fig. 251.—A case of true dextrocardia-anteroposterior view. There is a complete transposition of the viscera.

4. In pleural effusion or large empyema.
5. Pneumothorax (Figs. 257, 258).
6. In foreign bodies.
7. In pneumonia.
8. Postoperative massive atelectasis (Fig. 259).
9. Hernia of the diaphragm and eventration (Figs. 260, 261).
10. Scoliosis (Fig. 262).

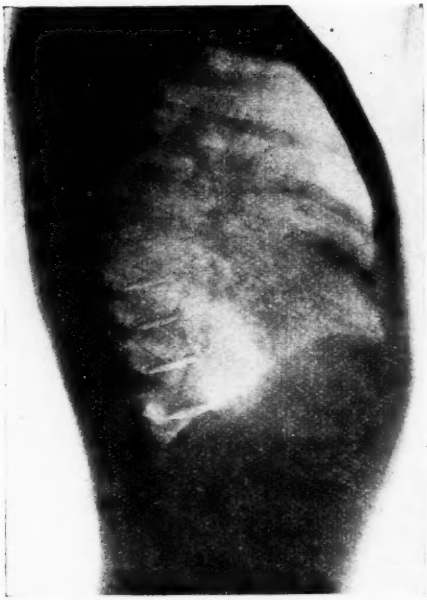

Fig. 252.—Same as Fig. 251. Lateral view of a true dextrocardia.

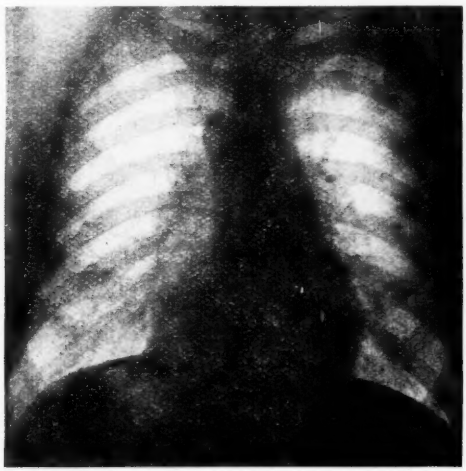

Fig. 253.—A case of pseudodextrocardia in a man aged forty-eight. There was no acquired reason for the displacement of the heart. There was no transposition of the abdominal viscera.

Fig. 254.—Complete dextrocardia in a case following influenza and an organized pleural exudate. The heart and mediastinal structures are entirely displaced.

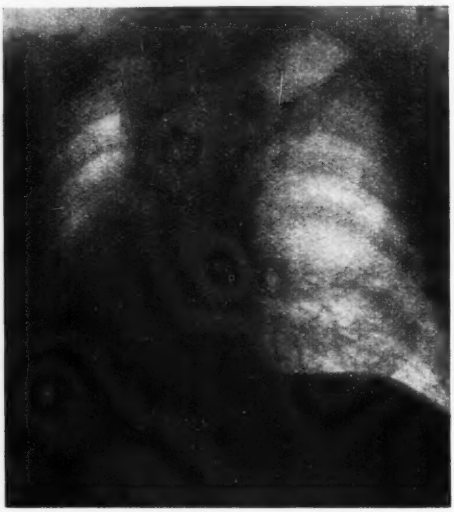

Fig. 255.—Man, aged fifty-five years, showing the displacement of the heart and mediastinal structures to the left, form an organized pleural exudate.

Fig. 256.—Lateral view of same patient seen in Fig. 255. The heart is displaced posteriorly by the organized pleural exudate. It is impossible to get this information from stereoscopic films.

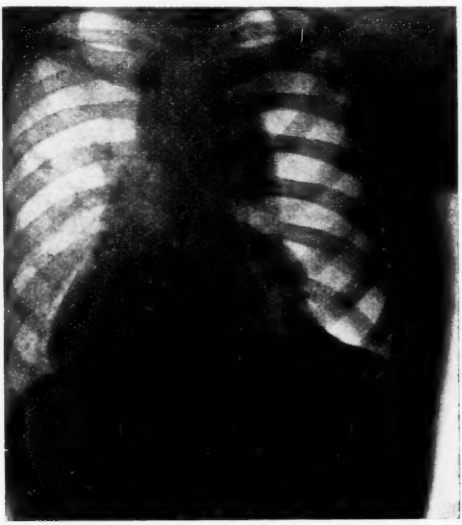

Fig. 257.—Slight sinistracardia following a complete spontaneous pneumothorax on the right side. The displacement of the heart laterally is usually very slight in pneumothorax on the right side. (See Fig. 258.)

Fig. 258.—Same patient as seen in Fig. 257. Lateral view shows displacement of the heart posteriorly. This is usually what happens in pneumothorax when it occurs on the right side, and possibly is one explanation of why there is so little sinistracardia in cases of right-sided pneumothorax. Compare Fig. 206.

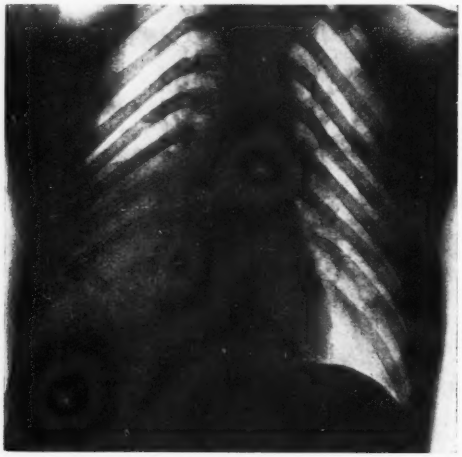

Fig. 259.—Postoperative massive atelectasis of the right lower lobe following an appendectomy. This examination was made in bed, but still shows slight displacement of the heart toward the affected side.

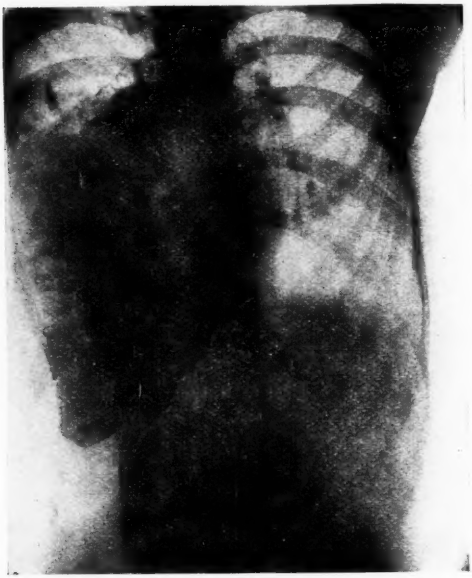

Fig. 260.—Slight dextrocardia with displacement of heart due to hernia of the diaphragm.

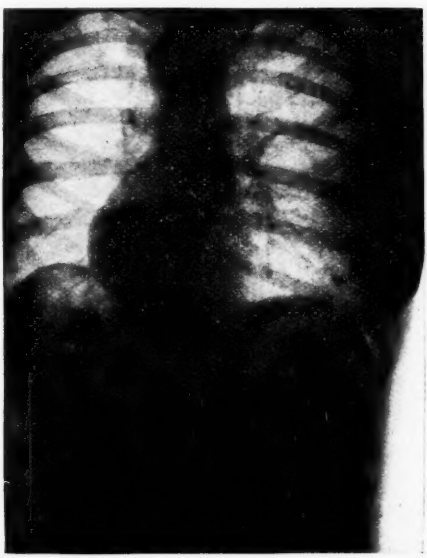

Fig. 261.—Eventration of the left diaphragm which displaces the heart slightly to the right.

The cases of true dextrocardia are rare and pseudodextrocardia is probably still more rare. True dextrocardia occurs in cases where there is a transposition of all the viscera. Pseudodextrocardia occurs in cases in which the heart is displaced but the abdominal organs are not.

Fibrosis following chronic conditions such as tuberculosis or organized pleural exudates will displace the heart toward the affected side by retraction.

Fig. 262.—Sinistrocardia in a case of scoliosis.

In pleural effusion and pneumothorax the heart is displaced away from the affected side.

The heart may be displaced in foreign bodies in the lower air passage. In those causing atelectasis of the lung the heart is displaced toward the affected side. When the foreign body is only partially obstructive and causes obstructive emphysema, the heart is displaced away from the affected side.

The heart is displaced slightly toward the affected side in

pneumonia and more so in postoperative massive atelectasis because of increased aëration of the opposite lung.

In hernia and eventration of the diaphragm, the heart is displaced away from the affected side because of increased height of the diaphragm in eventration and the abdominal viscera in hernia of the diaphragm.

In scoliosis, the heart is sometimes displaced because of the convexity of the spine (Fig. 262).

Tumors of the heart and pericarditis calculosa are not discussed in this Clinic because we have had no experience with either condition.

SICKLE-CELL ANEMIA

EDWARD STEINFIELD AND JOSEPH V. KLAUDER

THOUGH the discussion of an unusual case is open to the criticism of being more academic than practical, it may, however, serve some useful purpose. What is supposedly rare when first described becomes increasingly frequent as it is brought to our attention. On closer scrutiny a link between the rare and some commoner condition is noted, and in this way helps to complete our knowledge of certain groups of diseases. This thought in a measure pertains to the peculiar anemia of which our patient is an example.

The patient is a colored woman, thirty-eight years of age, who is referred by the Eye Department with the diagnosis of choroiditis. She was born in Huntington, Pennsylvania, and has lived only in that city and in Philadelphia. Her parents died of causes unknown to the patient, but she states that two sisters died of pulmonary tuberculosis. Two brothers are living and perfectly well. She was married at the age of twenty-one and at twenty-nine had a hysterectomy. She has had no children. At the age of sixteen she began to suffer from ulcerations about the lower legs, which would alternately heal up and break down, with the discharge of a thin purulent material. She has had aching in the muscles of the legs and has remained thin despite an ample and fairly balanced diet. She complains at present of weakness and dimness of vision. On inspection, you observe a moderate emaciation, a peculiar greenish color of the scleræ and pallor of the conjunctival mucous membrane. Note the scars of healed ulcerations about the lower legs, particularly the large annular scar over the left internal malleolus. These areas which on casual examination might be mistaken for healed gummatous or tuberculous lesions, have a more than passing interest

as you will see later. I will briefly summarize the results of the clinical and laboratory examinations.

Central Nervous.—No abnormal findings particularly important, no disturbance in vibratory sensation, sense of position, or tendon reflexes.

Endocrin.—No abnormalities of skeletal structure, facies, or hirsutism. Texture of skin normal, no areas of hyperpigmentation. Though eyes are prominent, there is no von Graefe's sign, no goiter, or other sign referable to the thyroid. Amenorrhea due to hysterectomy.

Respiratory.—Nose and throat examination: Tonsils small and buried; granulation tissue in posterior pharyngeal wall; roentgenograms of sinuses negative. Lungs: Clinical examination negative; Roentgen-ray examination reveals heavy peribronchial thickening with fine studding throughout each lung resembling effects of dust inhalation. No sputum was available for examination.

Cardiovascular.—Heart normal in size, no arhythmia, no murmurs, no atherosclerosis of radials, blood-pressure is 135/70.

Hematopoietic.—Slight cervical lymphadenopathy, but no enlargement of other nodes. Spleen is not palpable. The blood-count and other blood examinations are withheld for fuller discussion later.

Gastro-intestinal (Clinic of Dr. Bockus).—No masses or areas of tenderness or rigidity in abdomen; no hepatic enlargement. Fractional gastric: lowest reading, fasting, free HCl 0; total acid, 15; after fifteen minutes, free HCl 0; total acid, 10; highest at two hours, free HCl 25, total 40. Occult blood in first three tubes. Bile drainage: No A or C bile; 200 c.c. B bile; Giardia present. Feces: No occult blood; fatty acid crystals.

Urogenital.—Kidneys not palpable; pelvis characteristic of hysterectomy. Urine: Sp. gr. 1010, a trace of albumin, no sugar, no casts.

Blood examinations are as follows: Kolmer complement-fixation test for syphilis negative; Kahn reaction negative. Blood urea N., 16 mgs.; blood-sugar, 92 mgs.; uric acid, 5.5 mgs. per 100 c.c.

Van den Bergh: Direct reaction negative; indirect, 4.5 units.

Coagulation time of blood taken from vein eight minutes.

Volume index is 0.8.

Auto-agglutination of red cells negative.

Blood-counts.—September 24, 1926: Red blood-cells, 2,300,000; white blood-cells, 14,800; hemoglobin 43 per cent.

Differential: Polynuclear neutrophils, 63 per cent.; lymphocytes, 33 per cent.; transitionals, 3 per cent.; eosinophils, 1 per cent.

October 8, 1926: Red blood-cells, 2,050,000; white blood-cells, 25,000; hemoglobin, 40 per cent.

Differential: Polynuclear neutrophils, 69 per cent.; lymphocytes, 23 per cent.; transitionals, 2 per cent.; basophils, 3 per cent.; myelocytes, 3 per cent.

Many sickle-shaped red cells are present and about 15 normoblasts are seen while counting 100 leukocytes. Reticulocytes are approximately 10 per cent.

If you will now look at this blood slide under the microscope you will observe that nearly one-half of the red cells are distorted in a remarkable manner, that is, in the form of crescents and sickles. There are quite a number of normoblasts and an occasional myelocyte. There are no macrocytes or megaloblasts, which are characteristic cells in pernicious anemia. As you have surmised we have in this patient an example of sickle-cell anemia, reports of which are becoming more numerous each year. It was at first thought to be extremely rare, but many instances have been observed, particularly in the South. However, many of these are latent cases, whereas the number of active cases reported is not large. It is a singular fact, that though a large number of negro patients are seen in the Polyclinic Hospital, we have either apparently overlooked this condition or it is quite rare. Since it has been proved that the anemia is familial and hereditary, being found in the male and female as well as transmitted through either sex, there is no apparent reason for its greater prevalence in any particular district with the exception of the relative proportion of negro population. Some added factor of infection in the presence of

an inherited peculiarity of the hematopoietic system might cause differences in its incidence. The disease so far has been described in the negro race only. Its occurrence in individuals of disputed or doubtful negro descent may occasionally be of some medicolegal importance.

The condition is characterized by recurrent attacks of illness, during which the patient complains of moderate or severe pain and stiffness in the muscles and joints and occasionally

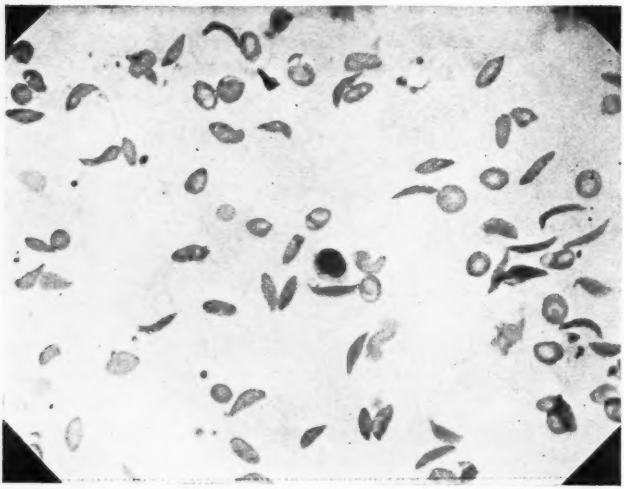

Fig. 263.—Characteristic sickle cells; a nucleated red cell is noted in the center of the field.

pain in the epigastrium or left hypochondrium. Weakness with or without loss of weight, fever, and a general moderate lymphadenopathy are described. However, the most characteristic physical sign is the greenish color of the scleræ. This is well brought out by the patient under discussion. Ulcers are frequently noted on the lower legs; the discussion of these lesions will be taken up by Dr. Klauder. Dyspnea and edema of the ankles may be noticeable, particularly if the anemia is marked. A moderate fever is present during periods of relapse. It has

been stated by some observers that the urine is of a low and fixed specific gravity. In this connection it is important to recall the observations of Christian upon the low and fixed specific gravity of the urine in many cases of severe anemia. This condition is likely to parallel the severity of the anemia and is not necessarily an indication of impaired renal function. A low gastric acidity, or even achlorhydria has been described. There is a moderate, or occasionally a severe, anemia, though the red cell counts do not usually fall below 1,500,000; the hemoglobin frequently varies from 30 to 60 per cent. The color index is normal or very little above normal. A leukocytosis is the rule and may reach a count of 15,000 to 20,000 with a normal proportion of polynuclear neutrophils. The diagnosis is made entirely upon the appearance of the red cells. In an active case, the changes are noted in the ordinary dried and stained blood spread such as made in this instance. In the latent cases the sickle cells are rendered apparent by placing a cover-slip over the fresh drop of blood on a slide and sealing the edges with petrolatum. In such preparations as high as 75 to 90 per cent. of the red cells may take on the sickle appearance in twelve to twenty-four hours. The phenomenon is supposedly due to some alteration of the cell wall or abnormal osmotic tension within the cell. It is not due to a peculiarity of the serum, since normal cells within serum from these patients do not undergo this change. The development of the sickle forms does not appear to a considerable degree in hanging-drop preparations; this suggests that a difference in the surface tension has accelerated the process. Reticulocytes are usually increased and may constitute 10 to 35 per cent. of the red cells. Many normoblasts are present and a moderate polychromasia is usual. Megaloblasts and macrocytes are rarely seen. Macrophages containing red cells are described, but were not detected in the present case. Myelocytes are present in small numbers. Auto-agglutination of the patient's blood-cells and serum *in vitro* has been observed, but was not detected with the blood of this patient. The anemia is associated with blood destruction, probably in the reticulo-endothelial system, and as a consequence bili-

rubin in the blood is increased with moderately high indirect van den Bergh reactions. Of the various anemias it seems more nearly related to family icterus, particularly of the acquired (Hayem-Widal) type. However, several notable differences are at once apparent. In family icterus, the splenic enlargement is constant, there are no sickle forms, and the lessened resistance of the red cells to hypotonic salt solution is a constant phenomenon. The evidence on fragility of the red cells in sickle-cell anemia is, I believe, somewhat equivocal; both increased and decreased fragility have been reported. The conflicting statements may be due to the proportion of sickle cells, inasmuch as Mason[1] has reported that these forms were more resistant than the apparently normal erythrocytes. However, the fragility in hemolytic icterus may have something in common with the sickle-cell changes, both being presumably due to alterations in osmotic tension in the cell, though manifested in a different manner. The points of resemblance are family incidence, a hemolytic type of anemia, a high reticulocyte count, and many normoblasts.

Pernicious anemia differs in many important respects. The red cell count reaches lower levels, and the color index is uniformly above one, except in marked remissions when approximately normal counts may be observed. The red cells are larger and the volume index in consequence is above 1.00. Megaloblasts are characteristic, though the number of nucleated red cells is less than in sickle-cell anemia. Leukopenia is usually present and becomes more pronounced as the regenerative power of the bone-marrow wanes. The achlorhydria and the spinal cord changes are distinctive. Despite remissions of possible long duration, practically all patients succumb to the disease and reports of complete recovery are excessively rare. It is rare in the negro race. The prognosis in sickle-cell anemia is poor in the active cases, simply because of the likelihood of intercurrent infections; the patient does not die from the severity of the anemia *per se*. The latent cases may be in apparent health and are likely to be discovered because of investigation of the family of an active case.

The treatment resolves itself into general supportive measures, such as a diet with a high protein and vitamin content, tonics, and hematinics. Splenectomy has been recommended for the active cases and would appear to be applicable to patients with increased fragility of the red cells.

DR. KLAUDER: Leg ulcer is one of the common symptoms of sickle-cell anemia; its occurrence as an expression of a blood dyscrasia is altogether unique, the relation is inexplicable. It has been present in all of the reported active cases in adults. It may appear, although exceptional, in early childhood. The ulcer, or its scar, is invariably present in patients seen in adolescence. It may antedate for years cardinal symptoms of the disease and may be seen in latent cases, or it may appear subsequent to the development of frank clinical symptoms of the disease.

Although it has been observed on various parts of the leg, and also on the dorsum, the ankle appears to be its most common site. It may be single or multiple, unilateral or bilateral, and is usually about the size of a quarter. Trauma may provoke its appearance. It presents itself as a discrete, sharply marginated, round or oval "punched out" ulceration, with a suppurating floor. In this regard it is comparable to a syphilitic ulcer involving the leg. It is amenable to cure by palliative measures. However, ulceration may reappear spontaneously, or following trauma at the same site, or it may develop at another site. No significant organisms have so far been demonstrated in the lesions.

The resulting scar resembles syphilitic scarring. It is atrophic, smooth, glossy, pliable, apigmented, and surrounded by a pigmented areola.

The patient whose photograph is shown in Fig. 264 is the one whose case record is herein discussed. She was thirty-eight years of age. The leg ulcer first appeared at the age of sixteen. Subsequently other ulcers appeared. These alternately healed and reappeared until about the age of thirty-four. The photograph shows the scars of such ulceration.

The diagnosis of leg ulcer associated with sickle-cell anemia,

based solely on the clinical features of the ulcer, is difficult and may be impossible. Indeed, such diagnosis is made from the constitutional symptoms and blood examination. It is important to bear in mind the possibility of sickle-cell anemia in dealing with leg ulcer, particularly in young negro patients. The leg ulcer may be the means leading to the diagnosis of sickle-cell

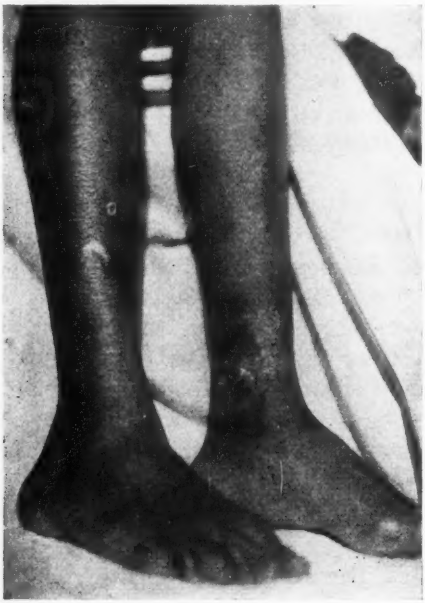

Fig. 264.—Scars of leg ulcer of sickle-cell anemia. Photograph of patient whose case record is herein reported. Leg ulcer is frequently present in patients with sickle-cell anemia. The ankle is a common site. It may be single or multiple, unilateral or bilateral, and is "punched out" in appearance. The resulting scar is atrophic, glossy, pliable, with a pigmented areola resembling syphilitic scarring.

anemia, since it may be the chief complaint of the patient, and may appear in latent cases or antedate frank subjective symptoms.

In differential diagnosis one should consider the common leg ulcers appearing in adolescence. These concern tuberculous

ulcerations—erythema induratum (Bazin's disease) and scrofuloderma; syphilitic ulcerations—the gummatous syphiloderm or gumma, and traumatic ulcerations.

Erythema induratum is seen usually in females under the age of twenty, although it is sometimes seen in middle-aged women. It is rare in males. The patients are usually tuber-

Fig. 265.—Tuberculous ulceration—erythema induratum (Bazin's disease). The calves of female patients under twenty are usually involved. Hard, painless nodules develop which slowly enlarge and ulcerate. The ulcers have suppurating floors and ragged edges. Scars of nodules which have involuted without ulceration are also shown. The process is very chronic.

culous and have a sluggish circulation, evidenced by a dusky hue of the extremities, the so-called chilblain circulation. In the majority of cases the disease is confined to the legs, most frequently in the lower part of the calf; at times on the anterior surface, and exceptionally, on the thighs and arms. The disease begins with single or multiple pea- to nut-sized, hard, painless

nodules beneath the skin. These slowly enlarge, the skin over them assuming a dull red or violaceous hue. In the course of some months the nodules may involute without ulceration, leaving a depressed atrophic area with some pigmentation. However, more frequently the nodules soften, sinuses form, discharging a seropurulent fluid, and later ulcers appear of varying size and extent. The ulcers have a suppurating floor and ragged edges. As a rule, the affection is painless. The course is chronic; partial or complete healing of the ulcers may take place and later relapse. New nodules may appear from time to time, some involuting, others ulcerating. The course may be prolonged for two or three years before terminating.

Figure 265 shows ulcerations of erythema induratum appearing on the calves and also scars of nodules which involuted without ulcerating.

Scrofuloderma (tuberculosa gummosa; tuberculosis colliquativa cutanea) is more frequently seen in children than in adults, and in them the typical and commonest form is seen as ulceration of the skin overlying tuberculous glands, bones, or joints. Another type is that described as scrofulous gumma, which is a circumscribed nodular gumma-like infiltration of the subcutaneous tissue not uncommon on the extremities. The condition usually begins insidiously with an indolent infiltration, or with one or more deep-seated nodules in the hypoderm or as a conglomerate mass. These may remain as such for weeks, causing little pain. There is a gradual enlargement of the deep-seated lesions; the skin becomes adherent, painful, and assumes a livid red or purplish hue. Ulceration slowly ensues. The ulcers have thin, ragged, undermined edges with a livid periphery. The floor is granular, which discharges a tenacious pus, perhaps blood tinged. It is well to remember that when a healing of a furuncle is unduly delayed, one should be suspicious of a tuberculous process.

The course of the process is slow and painful. The scrofulous gumma, both in the nodular and ulcerating stage, often bears considerable resemblance to the gumma of syphilis; but the latter runs a much more rapid course and forms round,

sharp-cut ulcers, with a scantier discharge; the periphery is harder and not inflammatory. The color of the scrofulous gumma is a livid cyanotic blue, whereas syphilis has a reddish, rusty color. The scars are different in tuberculosis. There is a thicker, heavy, at times keloid-like scar, while in syphilis the

Fig. 266.—Showing tuberculous ulceration—scrofulous gumma and scars of healed ulcers. This ulceration begins as a circumscribed nodular gumma-like infiltration of the subcutaneous tissue which slowly ulcerates. The ulcers have thin, ragged, undermined edges with a livid periphery; the floor is granular which discharges a tenacious pus. The thickened keloid-like scar shown in right leg is characteristic of this type of ulceration.

scar is thin, glistening, pliable, almost transparent, with a pigmented areola.

Figure 266 shows ulcerations and scars of scrofulous gumma in an adult. The characteristic hard, keloid-like scar is shown on the right leg.

Syphilitic leg ulcers, occurring in adolescence, may be lesions of either acquired or congenital syphilis. In acquired syphilis the ulceration will more likely be lesions of the secondary or late secondary stage, rather than the tertiary stage.

In patients in poor general health, as a symptom of so-called precocious syphilis, syphilitic ulcerative lesions may appear, ex-

Fig. 267.—Showing scars of multiple syphilitic ulcers. Multiple, bilateral syphilitic ulcers are lesions of late secondary or early tertiary syphilis; usually a symptom of precocious syphilis. Such lesions begin as pustules or papules, which ulcerate and become crusted, so-called rupia. The renal shaped, thin, pliable scars, with a pigmented areola, as shown in the photograph, are characteristic of the scar of syphilitic ulceration.

ceptionally in the secondary stage, more likely later from six months to a year of the infection. The condition may start as pustules, or as flat papules or nodules, and later as pustules which become crusted, beneath which ulcers form of varying depth. In some cases these crusts are laminated oyster-shell like, covering sharp-cut at times, quite deep ulcers. This lesion

is known as rupia. The resulting scar is smooth, round or oval, and more or less pigmented, especially about its borders. Such lesions are abundant on both legs and invariably associated with similar lesions on the body. The eruption is more scanty and not as generalized as are lesions of the secondary eruption. The course is slow, new lesions appearing at intervals, the process continuing for many months or more than a year. The scars of such ulcerations are shown in Fig. 267.

Fig. 268.—Syphilitic leg ulcer—an ulcerating gumma, a lesion of tertiary syphilis. The leg, the upper part, is a common site of a gumma. It is unilateral, solitary, and infiltrated. When ulceration takes place, it appears as a sharp-cut, "punched-out" ulcer which may be quite deep. It is non-painful, and of low-grade inflammation.

The gummatous syphiloderm or gumma is usually seen in the late or tertiary stage of syphilis as a solitary lesion. It may occur, however, in the late secondary stage as a symptom of precocious syphilis, about one year after the infection, in which event it is likely to be multiple.

It begins as a small solitary hard nodule, or a group of nodules in the hypoderm. As it enlarges it becomes adherent to the overlying skin, which becomes violaceous and slightly elevated. Its progress is rather rapid; it may attain the size of a small egg in about six weeks. The skin becomes taut, finally ulcerates, discharging a viscid bloody fluid and soon appears as a sharp-cut, "punched-out" ulcer which may be quite deep. Such ulceration is shown in Fig. 268. This ulcer may continue for years, slowly extending in depth and circumference.

Fig. 269.—Ulcer following trauma; secondarily infected. Such ulcers are acutely inflammatory. They are usually superficial and have a suppurating floor and irregular edges. They are painful, tender, and surrounded by an inflammatory zone.

The most frequent site of the gumma is the legs, usually the upper two-thirds, but it may occur on any portion of the body. The character of the scar resulting from healed syphilitic ulceration has already been discussed.

The gumma may occur at a late period in congenital syphilis, although less frequent than in acquired syphilis. As seen cutaneously, it does not differ from the lesion of the acquired form.

Traumatic ulcer of the leg, incident to trauma and secondary infection is acutely inflammatory, surrounded by an area of cellulitis. It is usually a solitary, superficial ulceration with irregular ragged edges, painful and tender. The base is suppurative, or covered with a yellowish membrane. A traumatic ulcer of the leg is shown in Fig. 269.

Fig. 270.—Chronic leg ulcer in adult, so-called "varicose ulcer." Invariably associated with varicose veins and sometimes with eczema. The skin of the leg is atrophic, thin and glossy, and readily breaks down following trauma. The ulcer, commonly located on the anterior surface of the lower leg, is superficial, sharply marginated, and accompanied by considerable pain.

The chronic leg ulcer, a frequent complication of eczema of the legs, or incident to varicose veins, the so-called "varicose ulcer" such as is shown in Fig. 270, is rarely seen in adolescence, and need not therefore be discussed in differential diagnosis.

The differential diagnosis of leg ulcer of sickle-cell anemia particularly concerns, therefore, differentiation from tuberculous ulcerations and those of late secondary syphilis, rather than the characteristic solitary ulcer of tertiary syphilis.

Note.—Acknowledgments are made to Dr. Boerner for personal attention to most of the blood work done in Dr. Kolmer's laboratory.

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GENERAL PARALYSIS OF THE INSANE: REMARKS ON THE DIAGNOSIS AND TREATMENT[1]

GEORGE WILSON AND N. W. WINKELMAN

WHEN a diagnosis of paresis is made it is well understood that the disease cannot exist without a previous syphilitic infection, and yet in the literature as recently as twenty years ago one reads of statistics "proving" that paresis is not due to syphilis. Krafft-Ebing's remarkable human experiment endeavored to settle the question as to the etiology of paresis. He selected 9 paretics in whom neither in the history nor in the physical examination was it possible to obtain evidence of syphilis. He vaccinated these patients both with the exudate from the initial lesion and the secretion from a mucous patch. The patients were kept under continuous observation for eighty days and not one of them reacted to the vaccination. He thus showed that in all of these patients there existed an apparent immunity against syphilis, and since it was known that immunity could exist only in the presence of a previously contracted syphilis it was believed proved that syphilis was present in these cases.

The discovery of the spirochete by Schaudinn and its presence in the brain in paresis by Noguchi were the last links in the evidence that "no syphilis . . . no paresis."

Paresis is a chronic inflammation of the brain and its coverings—a chronic meningo-encephalitis the pathologic picture of which is distinctive. One rarely is in doubt when there is found under the microscope a much thickened and fibrous pia with moderate round cells of the lymphocytic and plasma type in its deeper layers, distinct cortical atrophy due to loss of ganglion-

[1] From the Neurologic Department of the Medical School of the University of Pennsylvania, the Psychiatric and Neurologic Services and Laboratory of Neuropathology of the Philadelphia General Hospital.

cells with resultant distortion of the cortical layers, the presence of perivascular infiltration with the same type of cells noted in the meninges, and the finding of new vessel formation and rod cells.

Clinically paresis may cloak itself in so many different forms that it should always be ruled out in any mental manifestation, either psychotic or psychoneurotic occurring at any age, but es-

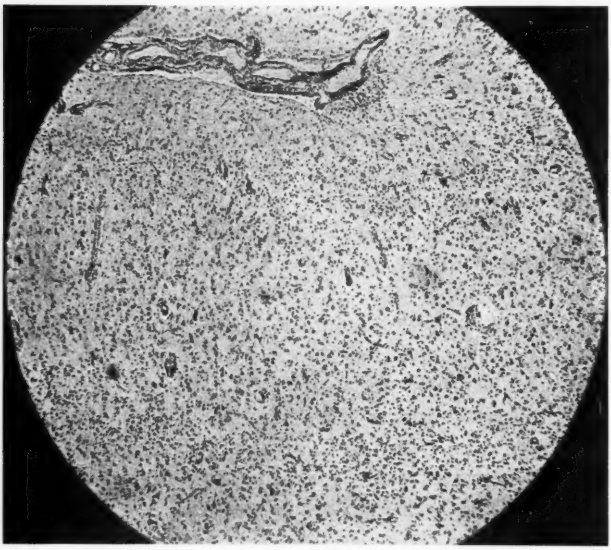

Fig. 271.—Paresis—moderately severe showing meningeal infiltration, cortical upheaval, and perivascular infiltration.

pecially between thirty and fifty-one. In the ordinary teaching and in most text-books of the day the grandiose form of paresis is stressed too greatly. There are still some who believe that paresis should not be diagnosed until the patient has delusions of grandeur and dementia. This is comparable to diagnosing cancer of the stomach when coffee-ground vomiting occurs or carcinoma of the rectum after metastasis has taken place to the spine. A careful record, especially one obtained from the family

of a paretic, will usually reveal that the individual case had shown psychic abnormalities for months or years before the disease became noticeable to all. The symptoms of early paresis may simulate the psychoneurotic reactions, and frequently they suggest other forms of mental disease, especially those characterized by excitement or depression. Commonly enough physical

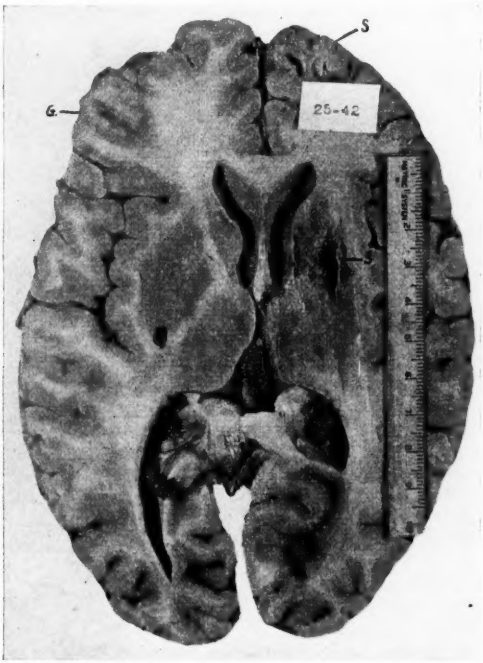

Fig. 272.—Brain in horizontal section showing gumma at *G* and softening at *S*.

manifestations occur at the onset, such as convulsions, attacks of transitory hemiplegia or monoplegia, confusion, amnesia, or aphasia. The physical findings, *i. e.*, abnormalities of the pupils, especially of the Argyll-Robertson variety, tremors of the face, tongue, and lips with the resultant speech disorder, increase of the deep reflexes, are so universally known that mention of them

is all that is necessary. Occasionally paresis is complicated by tabes; in that event the deep reflexes are lost.

Too much emphasis cannot be placed upon the value of the laboratory findings as an aid to the diagnosis. We have come to rely upon this help so much that it is difficult to place an overvaluation on it. In the untreated case of general paresis the blood Wassermann is positive and generally strongly so in over 90 per cent. of cases. The spinal fluid Wassermann is positive

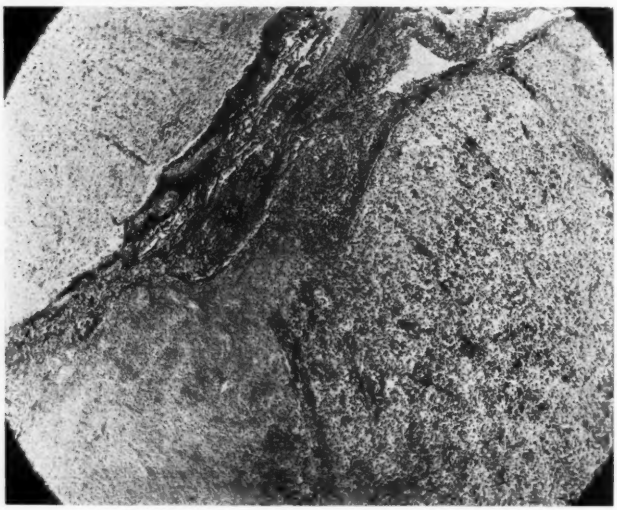

Fig. 273.—Syphilitic meningo-encephalitis. Note the marked subarachnoid accumulation of round cells and penetration into the cortex.

even in weak dilutions in what amounts to 100 per cent. of instances. The cells are increased, averaging in our experience between 30 and 40 to the cm.; they are of the lymphocytic type. The amount of globulin in the fluid is increased and the colloidal gold curve is of the paretic type, the typical curve reading 5555543210.

We believe that one of the most common errors that is made in interpreting laboratory tests on the spinal fluid is to place too great emphasis upon the value of the colloidal gold curve when

there is no other laboratory evidence of syphilis. It is well known that minor alterations in the curve occur in many organic affections of the nervous system and that often a paretic curve may be encountered in multiple sclerosis. In this disease, as in other non-syphilitic affections, the Wassermann reaction, the cells, and the globulin do not deviate from the normal.

Our attention has been called in the last few years to a tendency to disregard negative blood and spinal fluid findings in making a diagnosis of general paresis. While we cannot help but admire audacity we deprecate the lack of good judgment along these lines. In our experience with pathologic material in the laboratory of the Philadelphia General Hospital we have seen no untreated case with negative findings correctly diagnosed general paresis. We have also been impressed with the frequency with which some diagnose paresis when other conditions are more likely. This is seen especially in those whose relative inexperience in neuropsychiatry makes them rely to an unusual degree on the laboratory for aid in the diagnosis of paresis. Certain of the French neurologists believe that the high percentage of "cures" in paresis reported by Americans is explainable on the basis that many cases of cerebral syphilis are incorrectly diagnosed general paralysis of the insane. The prognosis in such cases being relatively good, the "cure" of general paresis therefore is not unusual. To illustrate these points we wish herein to report the following 12 cases. It will be noted that in some the laboratory findings were extremely suggestive, but pathologically none of them were examples of general paresis.

Case I.—A white man, aged sixty-five, developed epileptic fits fifteen years before his admission to the Philadelphia General Hospital on December 17, 1925; he had had 18 epileptic fits in the few hours preceding his admission to the hospital which he entered in coma. The right eye was absent, the left pupil was rigid. The reflexes were generally increased and plantar stimulation produced extension of the great toe on both sides. Laboratory examination: The blood and spinal fluid Wassermanns were negative. There were no cells and no globulin in the spinal

fluid, and the gold curve was 223432200. Microscopic diagnosis in this case was arteriosclerosis chiefly of the small vessels.

The history of epileptic fits dating back fifteen years and the negative laboratory findings, with the exception of the so-called syphilitic gold curve, certainly should have made one careful in diagnosing neurosyphilis, especially paresis.

Case II.—A white male, aged sixty, was admitted to the Philadelphia General Hospital December 22, 1925, and died in six days. Two years before admission he had a number of "weak spells," in which his feet became numb and he could not walk. He recovered and was well until five days before admission when the symptoms recurred and he also had difficulty in speaking; he became boisterous and profane and threatened to kill his wife, who stated that the patient had had considerable means and recently began to spend money foolishly. He often wandered away from home and got lost. The general examination was negative except for dysarthria. His memory was poor, but he was happy and oriented when questioned. Laboratory examinations: The blood and spinal fluid Wassermanns were negative; 8 cells and a trace of globulin were present in the fluid and the gold curve was 0001210000. Microscopic examination of the brain showed marked arteriosclerosis with multiple areas of softening and many small hemorrhages.

The age of the patient (we are fully aware that paresis can occur in the seventh and eighth decades, but it is uncommon) combined with negative laboratory findings in this case in our estimation should have been enough to rule out paresis. The tendency to diagnose paresis in the absence of positive laboratory findings practically always leads to a faulty conclusion.

Case III.—A white male, aged sixty-one, admitted to the Philadelphia General Hospital on May 20, 1925, and died six months later. Six months before his admission he fell to the floor one morning while dressing. He arose to his feet with difficulty and in one-half hour was able to walk. Three months later he had a similar attack, and after that he had about 6

more falls due to sudden giving away of his legs. Recently the left side of his body had become weak. Three weeks before admission he lost control of his bowels, and he had a recurrence of this unfortunate trouble twice. He had a chancre at nineteen followed by a sore throat for which he had little treatment. He drank to excess. The pupils were small, irregular, and reacted sluggishly; opthalmoscopic examination revealed blurring of the nerve heads and contraction of the vessels. The deep reflexes were increased. He talked of much money and said he owned oil lands; he was demented. The blood-pressure was 200/100. He developed cellulitis of the right elbow and died. The blood and spinal fluid Wassermanns were negative. The spinal fluid had a trace of globulin, 2 cells, and a colloidal gold curve of 0012211000. Microscopically the case was one of cerebral arteriosclerosis with areas of softening. The spinal cord was not obtained.

The mental picture in this case was not out of keeping with the diagnosis of paresis, and yet the diagnostician who relies to some extent upon the laboratory findings would never have made this diagnosis because of the absence of evidences of syphilis. It is this sort of case in which the laboratory findings are of overwhelming importance. This man had delusions of grandeur and dementia, and yet he did not have general paresis.

Case IV.—A colored woman of unknown age was admitted to the Philadelphia General Hospital March 13, 1926 and died in twelve days. She had been ill for two weeks with a cold and generalized aches and pains. She grew progressively weaker and for the last week was confused mentally. The patient was a thin woman of about twenty-five who lay in bed with her knees drawn up at right angles; the entire body was described as being spastic. The pupils were unequal and did not react to light or in accommodation. The face was expressionless and she was catatonic. She saw snakes and animals and heard voices, and was completely disoriented for a time. There was no record of alcoholism in this case. Laboratory examinations: The blood Wassermann was 4 plus, the spinal fluid Wassermann

was negative. The spinal fluid contained no cells and no globulin, and the colloidal gold curve was 1223331000. The microscopic diagnosis was pellagra.

Case V.—A colored woman of forty was admitted to the Philadelphia General Hospital April 24, 1926 and died in seven days. Three weeks before admission she had a carbuncle on the back of the neck. Two weeks later she developed erysipelas and shortly thereafter became irrational and unmanageable. On her admission to the Philadelphia General Hospital she was stuporous; the right pupil was larger than the left and was fixed, the left responded slightly to light. The neck was rigid and the deep reflexes were generally depressed, with an absence of the left knee-jerk. The left side of the face twitched during the last few days of her life. The eye-grounds were normal. Laboratory examinations: The blood Wassermann was 4 plus in the cholesterinized antigen and negative in the Noguchi. The spinal fluid was negative in every detal with the exception of a very mild alteration in the colloidal gold test which was as follows: 0001100000. Pathologically a small glioma of the right central ganglia was found.

Case VI.—A white female, aged fifty-six, was admitted to the Philadelphia General Hospital June 9, 1923 and died April 23, 1924. Her chief complaints were difficulty in walking and inability to retain the urine; these symptoms had been present for five years. She was stone deaf so that it was impossible to get a good history. The pupils were small and rigid to light. She was unable to walk and was barely able to stand with support; the reflexes were increased with a bilateral clonus and Babinski. Tests for sensation were impossible because of the patient's deafness, although it is likely that there was no loss of pain sense because of her reaction to stimuli. She showed no mental symptom except dulness. Laboratory examinations: The blood and spinal fluid Wassermanns were 4 plus in both antigens. The fluid showed a heavy trace of globulin, 5 cells, and the colloidal gold curve was 5555555543. A second examination of the

spinal fluid six months later showed exactly the same gold curve; the Wassermann was 4 plus in both antigens and a heavy trace of globulin was present. Pathologically the spinal cord showed a hypertrophic pachymeningitis in the cervical region. The cortex showed no evidence of paresis.

The diagnosis of paresis in this case was certainly not justifiable in view of the absence of mental symptoms and the presence of distinct evidence of gross disease of the spinal cord, which in itself if syphilitic in origin could give laboratory findings identical with those found in this case.

Case VII.—A black female, aged fifty-eight, was admitted to the Philadelphia General Hospital May 16, 1923 and died one year later. She was well until five months before admission when she suffered from attacks of chilliness and a feeling of general weakness. About a month after the onset she fainted and was unconscious for an hour; that same night she suffered from severe frontal headache and had difficulty in speaking. She complained of a girdle sensation in the upper abdomen, and also had great difficulty in walking. On examination the woman was found somewhat excitable and much undernourished. The pupils were irregular, unequal, and reacted sluggishly. The right optic nerve was normal, the left fundus showed two white spots, probably an exudate. A coarse tremor of the tongue was present, producing a speech difficulty. The reflexes on the left were increased without ankle-clonus or Babinski. A later and more detailed mental examination showed the patient to be quiet, not irritable, excitable, or depressed. She answered questions promptly and intelligently, although her speech was a little slow. She had no delusions, paranoid tendencies, hallucinations, or obsessions. She was completely oriented; her judgment was good and she had insight into her nervous condition. In April of 1924 she awoke one morning paralyzed on the right side and aphasic. After the stroke her memory was poor, she was disoriented and little that she said could be understood. After the stroke she lay with both her legs markedly flexed and yelled every time her extremities were moved. Her deep reflexes were

universally increased. Plantar stimulation produced no movement on the right and flexion on the left. At this time the diagnoses of general paresis and right hemiplegia were made. The laboratory findings showed 4 plus Wassermanns in both blood and spinal fluid. There were 383 cells and a faint trace of globulin in the spinal fluid and the following gold curve: 5555555432. Pathologically, the brain showed multiple areas of thrombotic softening in the cortex and basal ganglia and meningovascular syphilis, non-paretic in type.

In the consideration of this case it should be pointed out that the woman showed practically no mental symptoms until after she had suffered a stroke of apoplexy. Like quite a few other cases which we have seen, this one confirms our belief that given a gross organic disease of the brain or cord other than tabes paresis is not likely to complicate the picture.

Case VIII.—A colored woman of fifty-seven was admitted to the Philadelphia General Hospital on January 19, 1925 and was discharged three weeks later; she was readmitted in March, 1925 and died in two weeks. She had been well until four years previously when she developed frequency and incontinence of urine. The night before her first admission she became dizzy and had generalized twitchings which became continuous. She vomited while in the receiving ward. Examination showed the pupils were small, equal, and fixed. The septum was perforated. The extremities were rigid and the seat of twitchings; the reflexes were increased with an atypical Babinski on both sides. A few days after admission she had a convulsion which began by an extension and abduction of the right arm; the spasm then spread to the face which was drawn to the right in a series of rhythmic movements. The convulsion did not involve the right lower extremity nor the left side of the body. Two days later she came out of her coma, was belligerent, asked for whisky, and volunteered the information that she had "sick spells" with unconsciousness since birth. The eye-grounds were negative at the time of her first admission; the blood and spinal fluid Wassermanns were 4 plus in both antigens; spinal fluid showed no glob-

ulin, 3 cells, and the following colloidal gold curve: 4444442211. On her second admission she had a slurring, unintelligible speech. Her attention could not be held, she moaned whenever touched, but otherwise lay in bed quietly with the knees drawn up to the chin, paying no attention to her surroundings and being extremely non-co-operative. The deep reflexes on the right were somewhat increased, those on the left were diminished and plantar stimulation produced flexion on both sides. She became euphoric, thought that she had a great deal of money, and the record read that she was "hallucinated," although it gave no details. A few days later she became weaker and died. The later laboratory examination of the blood and spinal fluid showed a 4 plus Wassermann in both with a gold curve of 3555554221. Pathologically this case was extremely interesting; the brain showed a gumma of the left frontal lobe, and a second gumma in the left parietal region. Microscopically the diagnosis of neurosyphilis with multiple gummata and vascular softening of the right frontal lobe was made.

The diagnosis of paresis in this case was warranted from the mental, laboratory, and physical findings, yet it was incorrect. The mental symptoms were due to bilateral lesions of the frontal lobes and the laboratory findings were due to cerebral syphilis.

Case IX.—A colored female, aged sixty, was admitted to the Philadelphia General Hospital on February 13, 1924 and died six days later. She was taken ill three weeks before admission with loss of power in all four extremities and a disturbance of speech. Her son later said she had been ailing indefinitely for two years. She had marked loss of strength in her hands and could not pick up or hold objects easily. She was of low mentality and had a surly disposition, although when not disturbed she lay quietly in bed, said that Jesus often spoke to her, but she could not say what He said. She had no visual hallucinations. The pupils were irregular and did not react to light. The deep reflexes were about normal, and plantar stimulation produced flexion on both sides. A few days before she died she lost her speech, was semiconscious, and developed Cheyne-Stokes' res-

piration. The blood and spinal fluid Wassermanns were 4 plus: the spinal fluid contained 20 cells, a trace of globulin, and showed the following colloidal gold curve: 1123321000.

Pathologically the diagnosis in this case was cerebral arteriosclerosis, thrombosis of the superior longitudinal sinus, and a small area of softening in the pons. A mild syphilitic basilar meningitis was found.

Case X.—A white male, aged fifty-five, was admitted to the Philadelphia General Hospital on October 23, 1922 and died six days later. He was taken ill suddenly with paralysis of the right side and aphasia. The blood Wassermann was 4 plus in the cholesterinized antigen and negative in the Noguchi. The spinal fluid was 4 plus in both antigens, had 80 cells to the cm., a trace of globulin and the following colloidal gold curve: 5555543200. Necropsy showed thrombosis of the left middle cerebral artery with softening of the parts supplied by it. There was no evidence of paresis, but cerebral syphilis was easily detected.

Case XI.—A white male, aged fifty-six, was admitted to the Philadelphia General Hospital on February 20, 1926 and died in two weeks. Five weeks before admission he felt dizzy, had a headache and paresthesias on the left side. In the course of two weeks he recovered fully, but a few days later had a recurrence of his symptoms and became paralyzed on the left side and then had the ordinary signs of a left hemiplegia. The speech was thick; the pupils were unequal, irregular, and reacted slowly to light. He deteriorated mentally. Laboratory examinations: The blood and spinal fluid Wassermanns were 4 plus. The fluid contained 240 cells on one examination and 156 on another; a test for globulin was not done. The colloidal gold curve was 5544321100. Microscopically, arteriosclerosis and meningeal syphilis were found.

Case XII.—A white man, aged thirty-seven, was admitted to the Philadelphia General Hospital in coma with generalized convulsions, more pronounced on the right. The right lower extremity was paralyzed; the pupils were fixed and irregular.

Later he cleared up to some extent and was euphoric; he also had delusions of marital infidelity, having seen his wife having intercourse with another man. He heard his wife talking to him and said she was trying to control his mind. The man had been drinking to excess. He was disoriented and his memory was poor. Laboratory examinations: Blood Wassermann was 4 plus in both antigens. Spinal fluid: 4 plus in both antigens; cells 30 the first time and 240 the second; globulin positive; gold curve 1123340000 and 1123342210. The mental picture in this man, colored as it was by hallucinations and delusions of marital infidelity, with the euphoric state pointed more to alcoholism than a syphilitic mental disease. The colloidal gold curve was of the syphilitic type; there was no record of treatment. The pathologic diagnosis was cerebral syphilis, non-paretic in type.

While it may appear academic to some to differentiate cerebrospinal syphilis from paresis by "peering through a microscope," it is important from a therapeutic standpoint.

COMMENT WITH A NOTE ON TREATMENT

The first five of the foregoing cases present a variety of pathologic conditions, all of which were diagnosed general paresis during life. Three of the patients had cerebral arteriosclerosis; one had pellagra and the fifth had a brain tumor. Careful examination of the records will reveal that there was some justification from the clinical standpoint for making a diagnosis of general paralysis of the insane; however, when the laboratory aspect of these cases is approached one quickly appreciates the fact that there was little to corroborate the diagnosis of syphilis in any form, more particularly general paresis. The blood Wassermann was negative in 3 of the 5 cases and positive in the other 2, and as future events proved it was probably an incidental finding in these 2 patients. The spinal fluid, however, was normal in all 5 cases with the exception of an alteration in the colloidal gold test, usually of a mild degree.

We would again like to stress the point that in a case which has received no antisyphilitic treatment a change of any sort, even a paretic one, in the colloidal gold curve does not indicate

syphilis of the nervous system. We have frequently heard the subject discussed, and have been struck by the number of times that great emphasis has been placed upon alterations of the gold curve as indicative of neurosyphilis of some variety. As we pointed out earlier in our presentation, there are certain affections of the nervous system which commonly produce alterations in the gold curve; *e. g.*, multiple sclerosis as long as the case is progressing or is in an active stage will show an alteration of the curve, frequently of the paretic variety. In the diagnosis of general paresis, therefore, one must take note of negative laboratory findings, and when the laboratory does not give confirmatory evidence of the presence of syphilis the diagnosis of general paresis should not be persisted in, and we believe that it should be abandoned entirely.

The remainder of the cases in our series presents a problem in exact diagnosis and in prognosis. All of the 7 cases had syphilis of the nervous system, either of the brain or cord or of both, and some of them had mental symptoms which were strongly suggestive of general paresis; *e. g.*, in Case VIII the patient had fixed pupils, increased reflexes, Jacksonian convulsions, euphoria, and delusions of grandeur, in addition to which the blood and spinal fluid Wassermanns were 4 plus and the colloidal gold curve was of the paretic type. There would be few indeed who would fail to diagnose general paresis with such an array of signs and symptoms, yet the pathologic diagnosis was gumma of the left frontal lobe and left parietal lobe and a softening of the right frontal lobe.

This group of cases also brings up an important point, viz., that in the presence of evidence indicative of gross organic disease such as a softening, general paresis usually does not complicate the picture. The attacks of hemiplegia or monoplegia which occur in paresis usually clear up in a short time. If, however, the patient has a hemiplegia which comes on as the result of cerebral softening and the symptoms of which remain more or less fixed, such a patient usually has frank cerebral syphilis and not general paresis (*vid.* Cases VII, X, XI, XII). It is also true that gross disease of the spinal cord, with the exception of tabes

dorsalis, is usually not complicated by general paresis. In Case VI the symptoms and signs were strongly indicative of a spinal cord lesion rather than a cerebral one. The microscopic study of the central nervous system showed cervical hypertrophic pachymeningitis.

When patients with syphilis of the nervous system, especially the cerebral type, indulge in alcohol mental symptoms may be quickly produced, and the patient should not be labeled a general paretic until a proper period of observation has elasped. We realize, of course, that any patient who is psychotic may find refuge in alcohol. Cerebral syphilis, non-paretic in type, may in itself account for mental symptoms, and in such cases the differential diagnosis between paresis and cerebral syphilis is extremely difficult.

The treatment of paresis has been notoriously ineffectual, and the question is often raised: Is it worth while to treat a case which has been diagnosed general paralysis of the insane? The mere fact that the diagnosis itself is often incorrect and is frequently made when the patient is suffering from frank cerebral syphilis, would make him who is an extreme nihilist as far as therapeutics are concerned step in with his heaviest artillery. Care must be exerted, however, in reporting results, and it must be recognized that many cases of cerebral syphilis, especially if the patient is alcoholic, produce mental symptoms that are at times almost indistinguishable from those of general paresis. Undoubtedly, as the French assert, many of the "extraordinary" results obtained in this country in the treatment of paresis have been obtained in cerebral syphilis, non-paretic in type. As far as the actual treatment of paresis itself is concerned there is no doubt that the use of arsphenamin, neo-arsphenamin and sulpharsphenamin, mercury, and the iodids has produced no results worthy of note.

In tryparsamid, however, we have a drug, used originally in the treatment of African sleeping-sickness, the therapeutic results of which far surpass any of those mentioned above. The dose of tryparsamid is 3 grams dissolved in 20 c.c. sterile, distilled water, and given intravenously weekly for 8 doses. After

this a rest period of two to three weeks should be allowed, and then the whole procedure repeated. For practical purposes the only unhappy result which occurs after tryparsamid therapy is involvement of the optic nerves, hence the drug should be stopped immediately if the patient complains of visual phenomena.

The treatment of paresis by the production of fever has recently come into great vogue and numerous references in the literature are made to this form of therapy. The method most commonly used (and the best for the production of fever) is the inoculation of the patient with malaria which should always be of the tertian variety and non-malignant in type. Patients who receive this form of therapy should be physically sound as far as the lungs, heart, liver, arteries, and kidneys are concerned.

We have in preparation, in conjunction with Dr. Samuel B. Hadden, a paper on our results in the use of malaria in paresis, the results of which are comparable in every way and perhaps somewhat better than those which are obtained with tryparsamid.

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INDICAN: ITS PERIODICITY OF ELIMINATION[1]

GORDON J. SAXON

THE essential part of a history, in the investigation of any patient, is a statement concerning the colon function. This may be based on *x*-ray findings, but usually it is based on the patient's statement as to the frequency of bowel evacuations. When *x*-ray findings which show definite evidence of colonic stasis are compared with the patient's estimate of his colon function, surprising degrees of discrepancy will be found often to exist. The patient will state that the clock can be accurately set by the degree of regularity of his bowels or that he has two or three copious evacuations a day, and that no concern or apprehension need be had in his case. Patients of this description can usually disarm a physician of suspicion and an aggravated degree of colon retention goes on unobserved. It is the family physician and not the hospital specialist who has the first opportunity in this connection. The hospital specialist may easily fall into the same error as anyone else, but in his work grave conditions are encountered and not infrequently the *x*-ray examination reveals a colon retention which had not been suspected. In the case of the family physician, however, many patients present themselves with some of the minor manifestations or results of colon stasis, which in themselves are not sufficient to occasion an *x*-ray examination. The usual questions are asked about the "bowels," and the subject is often dismissed because of negative answers. Frequently, stasis is suspected as the cause of a symptom or group of symptoms and the therapeutic test is applied; a laxative is used to decide the issue, and if the symptoms disappear—as they may—it is safe to conclude that an intoxication has been

[1] From the Clinical Laboratory Service and Private Practice of Gordon J. Saxon. Read before the Staff of St. Luke's Hospital, Bethlehem, Pa.

the cause. But if the symptoms do not disappear, it is not safe nor correct to infer that an intoxication had not been operating, for here, again, the *x*-ray has frequently established that this evidence is not sufficient, that frequently repeated laxatives or even drastics may fail to remove all of the offending material from the colon. It is scarcely possible—as ideal as it might be—to have every ambulant patient with minor symptoms subjected to an *x*-ray investigation. How else may one determine whether colon function is normal, or, if abnormal, how it is responding to treatment? It is highly important that accurate information be obtainable in this respect, for if it be not had, a valuable plan of treatment might be abandoned simply because a group of symptoms had failed to disappear. It would not be so difficult to work understandingly where colon function is concerned, if the symptomatology were not so variable. This phase of the subject will be mentioned later.

In a previous communication (Indican—Its Periodicity of Elimination, Archives of Pathology and Laboratory Medicine, November, 1926, p. 796) the writer has called attention to the more or less tidal character of elimination of indican and that a correct evaluation of the clinical worth of the test for indican in the urine must premise that the test has been performed by accurate quantitative methods on the twenty-four-hour output, or that the simpler extraction colorimetric methods have been employed on specimens collected every three or four hours over an arbitrarily chosen period, preferably not less than twenty-four hours. As ordinarily done in hospitals, and in private practice, a single specimen of urine is tested, or a composite of the night and morning urine is used, or a composite is chosen from the twenty-four-hour output. By the first and second plans, one may miss the indican shower in from 15 to 85 per cent. of the instances where it has actually been present. By the third plan, the total indican output may give only a feeble color reaction under conditions of high dilution that may exist. It is easy to see, therefore, why with a patient in whom the *x*-ray has revealed a seventy-two hour colon retention and the solitary specimen sent to the laboratory shows no indican, the test in

question should fall into disrepute as an index of colonic retention and putrefaction. Much confusion has also lent discredit to this index, since the argument has been advanced by those in whose minds the chemistry of the situation has not been clear, that indican is not toxic and consequently is not concerned in the production of symptoms. Nothing could be nearer the truth. The effort is being made here, however, to present indican, when properly looked for, as a true criterion of the degree of putrefactive cleavage in contradistinction to digestive cleavage and not as a causative agent of any symptom. Where indican is present there also will be found all of its bad company, and combined with the toxic cleavage products is a proportionate number of exotoxins, the products of the bacterial agents of putrefaction themselves.

The routine for the collection of specimens and the performance of the test is best done by giving the patient a package of six 1-ounce corked bottles, and requesting that a bottle be filled each time urine is passed, throughout a period of twenty-four hours, giving instructions that the specimens be kept in a cool place during collection. The patient can supply an additional bottle or so if more than six urinations are had in the twenty-four hours. It is important that instructions be given about water-drinking during the collection. There are those who have become obsessed on the subject of water drinking and regard their kidneys as cheesecloths through which water may passively flow to wash the supposedly existing barnacles away. Such a plan would interfere with the test, and it must be seen to that no more fluids are taken than are dictated by thirst . . . that all-sufficient guide as to the amount of water to be consumed.

Half the content of each bottle may be used in making a composite for the routine urinary analysis. To each of the half-emptied bottles, add approximately $\frac{1}{2}$ ounce of U. S. P. chemically pure hydrochloric acid as well as 2 or 3 drops of a 1 per cent. solution of potassium permanganate. Then add 2 c.c. of chloroform. The bottles may now be agitated by a rocking motion by taking three in each hand. Care should be taken because so many patients have been fighting that bugaboo, acidosis, by

taking regularly large quantities of soda, and a disagreeable minor accident due to vigorous effervescence and popping of corks might ensue. If the test should be negative after the first agitation, set the bottles aside for two minutes and again mix the contents. If the chloroform layer darkens, the process should be repeated until the deepest color obtainable is imparted to it. If iodids should interfere with the typical indican color, render the iodid reaction colorless by the use of sodium thiosulphate. The use of the ordinary prescription 1-ounce bottle has the advantage that it serves as a test-tube and will stand by itself and make exhibition of the array less troublesome.

Patients become interested in obtaining what I have called a perfect "score," and devote themselves to their treatment the more energetically and the more intelligently if the test is performed for their inspection. In the beginning of treatment four of the six collections may contain a large amount of indican and two may have none. The gaming instinct is present in most of us, and an appeal is made by expressing such a result as *four* to *two* against the patient. Under appropriate treatment the gradual disappearance of indican from the urine is commensurate with the disappearance of the symptoms referable to colon stasis and may be complete, after a few days; but in aggravated cases, this goal may not be reached for from three months to two years, in which event none of the six collections may contain indican and the score is said to be *six* to *nothing* in favor of the patient.

The plan of treatment to pursue must naturally vary in each case, but I am going to present the more essential details of a non-medical management which has served my purpose well and has won the approval of many sufferers. In the presentation of it, my idea will be to do it, much as it is outlined to the patient, for his complete understanding and appreciation. The method is not advocated to the exclusion of others, for many individuals will not lend themselves to the scheme until they are in the throes of all the ill-consequences of colon intoxication.

Fulness is the normal stimulus for any hollow viscus and this applies with special force to the rectum. Fill the rectum with a

scant pint of water and by voluntary effort it can be expelled, if the attempt be made immediately after the introduction. By sufficiently prolonged voluntary effort the mechanism of defecation can usually be enhanced to its conclusion. But, if this should not result, repeating the process again, or still again, at the same sitting, will probably be attended with success. Lack of success, however, does not mean but that the colon contents may have been advanced by the induced peristalsis which takes place throughout the entire colon, thus facilitating matters for a subsequent attempt. It is better that the patient think of the procedure, not as an enema, but rather as a water suppository, as something which activates the mechanism of defecation and not as a flushing agent. The idea should be imparted that the water is to be introduced low, with a short nozzle, and not permitted to get into the upper colon. Hence, it is best that it be done while sitting on the toilet and not while in the recumbent posture, unless a high colon irrigation is the object. It should be explained that it is in the rectum that fulness should be brought about, for fulness higher up does not induce the mechanism of defecation any more than pressure in the pelvis of the kidney or in the ureter will induce the mechanism of urination; neither will irritation of the nasal mucous membrane induce gagging or vomiting, nor will irritation of the throat cause sneezing, and, if to repeat will lend force to the argument, let it again be said: to cause the onset of defecation and the reciprocally associated colonic activity, introduce a scant pint of water (more water causes distension and eventual weakening of the rectal muscle coats) into the rectum and see to it that by virtue of the sitting posture, gravity will prevent its going higher into the colon. Immediate and forceful expulsion of the water is advisable. To retain it with the idea that it may soften the rectal contents is wrong, for usually in these colon conditions, the accumulation is in the colon and not in the rectum. It should be pointed out to the patient that the water serves simply as something to have the first part of the bowel movement with in order that from this point on sufficiently prolonged and serious-minded voluntary effort may be productive. If the plan is used once,

twice, or even four times a day, depending upon the degree of indicanuria, the colon contents will not only be advanced at each seance, but the circular and longitudinal muscles, which do not come under voluntary control, will be exercised in a normal physiologic manner. At the same time, more copious defecation is effected with the result that the burden under which the liver has been laboring will be lessened and a vicious cycle will be broken, in this respect at least.

If it be explained to the patient that the blood going to the arms, legs, head, and trunk returns to the heart again, but that the blood sent to the abdominal viscera does not return to the heart directly but goes to the liver first, it will become obvious to him how the liver may be seriously damaged, and greater faithfulness to the treatment will be the result. I do not believe I have ever encountered a patient—school teacher, college professor, engineer, architect, clergyman, etc.—who has understood the difference between the venous return of the systemic circulation and that of the portal circulation.

As the indican in the urine decreases and the patient more and more approaches a perfect "score," the number of enemas or water suppositories may be decreased and sufficiently prolonged serious-minded voluntary effort substituted. Not infrequently normal colon tone and habituation can be established. As an accompanying feature of the treatment it is essential that every patient of this kind be advised against the use of sweets of all forms and also against the excessive use of starches. In those whose fuel requirements are high, fats can be substituted. In other words, the diet should contain sufficient protein, preferably in at least two periods, to meet the bodily requirements of repairs. To this add the truck vegetables, especially for their vitamin and mineral content, and allow sufficient starches, especially in lean individuals, in order that too drastic a dietary change may not be brought about. Allow bread and potatoes in moderate amounts or, if not these, some other starch, but never in the form of puddings or other combinations of starch and sugar. Butter and other fats should make up the caloric deficiency if such is found to exist. There is no doubt that a diet

that leans slightly to the ketogenic side is better borne than a high carbohydrate intake.

It is not infrequently seen in patients with colon retention, whose symptoms include vertigo, scintillating scotoma, migrainal type of headache, or urticaria, that these symptoms can be brought on almost at will by instituting a carbohydrate debauche. One of my professional friends, whom I call a "dessert hound," admits this to be the cause of many of his worst headaches.

The writer is opposed to the use of bran. If ruffage should prove in a given case to be required, it is better to use lettuce, celery, spinach, cabbage, cauliflower, etc., since these have a more favorable mineral and vitamin content than bran and are better borne. Diets designedly high in ruffage are not included in the treatment, for in the cases in which flaccidity of the colon exists ruffage increases the existing difficulties. Excess of ruffage may result in still further dilatation of the colon and an aggravation of an already well-established fundamental intoxication.

It is very essential that the patients be instructed to effect their treatments expeditiously, for if they do not do this there will develop a consciousness of effort which will render the plan irksome, in which case it will be abandoned. Teach them to take their small low enemas with the same lack of consciousness of effort with which they put on their shoes, care for their hair, or disrobe. If the plan is not successful in the beginning, it does not imply that responsiveness may not be induced by continued trial, and it may be advisable to supplement the scheme with the periodic use of laxatives, with the use of mineral oil, and also possibly some of the acidophilis preparations. In the use of the acid milks, however, care must be had, for not infrequently they are so stimulating to the hydrochloric acid producing mechanism that very disagreeable degrees of hyperchlorhydria are induced.

I hope I have not seemed to imply that no other plan of treatment should be used, nor that the plan here presented is presented in anything but the most incomplete and elementary manner. Those following me out must adapt the routine to the

patient's needs and their own conceptions, and include such valuable measures as exercise in the open air, exposure to the sunlight or ultraviolet rays, or any other helpful plan—the inclusion of which the brief scope of this paper has not permitted.

Symptoms are often not attributable to their proper cause in the condition under discussion, because they are so bizarre in type and varied in distribution. Some of the more important symptoms which have been seen to attend stasis will now be presented: vertigo, with or without tinnitus, or tinnitus alone, are frequently encountered, as is also scintillating scotoma, together with sensory disturbance along the distribution of the fifth nerve, and migrainal type of headache. Mental depression and delirium and mental states simulating paranoia may also be present. Lack of concentration, both as to intensity and as to length of time, is in some individuals an outstanding feature. I recall a busy gentleman, an architect, in whose personal employ was a score of workers, whose output depended largely upon his mental alertness. He consulted me concerning the advisability of getting out of business "while the getting was good." His power to concentrate had become so impaired that the morale of his office force was becoming seriously threatened.

Aside from a low-grade anemia nothing else was found except colon stasis. Three of his six specimens of urine gave an intense indican reaction; the remaining three were negative. This was found in spite of the fact that he claimed he had three normal bowel evacuations a day. He became somewhat incensed when first informed that his condition was probably due to colon stasis. Appropriate treatment soon restored him to perfect health, in which state he has remained for the past five years.

This last mentioned symptom may or may not be accompanied by mental irritability. Frequently, one sees a peculiar type of fear and apprehension in which the patients are not able to define the fear nor are they able to say what causes their apprehensiveness. One patient did not leave the house for a year unless accompanied by a member of her family, because mental lapses of infinitesimal duration caused her to fear that she might fall on the street. She had been treated

with bromids and sedatives, but the neurologist had failed to recognize the fundamental underlying condition of colon stasis. When this was cared for, the patient's health was restored.

Intercostal and lumbar neuritis, as pointed out by J. B. Carnett,[1] are often mistaken for appendiceal and other intra-abdominal conditions and the patient is occasionally operated upon with end-results that are anything but gratifying. His method of examination should be employed in the differential diagnosis of intra-abdominal and adjacent parietal conditions. Stomach manifestations, such as hyperchlorhydria or achylia gastrica, are frequently noted and are seen to disappear under appropriate colon treatment. No doubt there are many unexplained skin manifestations which are attributable to this cause, but as easily recognizable skin manifestations of it one often encounters urticaria and acne vulgaris. The latter is often associated with a high sugar intake, which has not only caused a hyperglycemia, but also an increased activity of the colon putrefactive processes. Care of the colon and adjustment of the diet are often followed by clearing up of this condition, as well as the more aggravated forms of skin infections such as furunculosis and carbunculosis. Intense redness of the palms of the hands, especially the hypothenar eminence, is an almost unfailing sign of colon intoxication.

Distention of the colon, especially in its transverse portion, frequently gives chest symptoms, such as shortness of breath and palpitation of the heart; the patient, not being able to lie down, paces the floor and is in an anxious state of mind. This is frequently encountered in those bran enthusiasts who in such an event take ever-increasing quantities of this by-product in the hope of getting relief from the condition for which the bran itself is responsible.

One symptom which is disagreeable for those about the sufferer is body odor and fetor of the breath. Those patients having their retention in the cecum, and especially if this is accompanied by incontinence of the ileocecal valve, have a fecal odor on the breath. When it is explained that this odor

[1] Surg., Gynec., and Obst., May, 1926, pp. 625–632.

is not necessarily an odor *like feces*, but is the odor *of feces*, and that it is brought about by absorbed volatile products from the colon being thrown off into the lungs and emanating from the breath, faithfulness to treatment is often stimulated, for the thought is not a happy one for the average patient to entertain. Retentions low in the colon are not so apt to be accompanied by fetor of the breath, and the tongue is often reddened in contradistinction to the badly coated tongue seen in cecal retentions.

The writer is not impressed with the importance of pressure per se in the colon as the cause of headache and other symptoms attending colon stasis. Alvarez contends that relief of pressure is attended so promptly by relief of symptoms that pressure itself is their cause. He further states that headache and similar symptoms can be brought about by packing the rectum with cotton. This may be true, since the establishment of obstruction anywhere in the intestinal tract will be attended with unusual symptoms; but not infrequently one has the opportunity to observe a patient who has been operated upon for prolapse of the rectum, in whom a canalized pack is used. In such instances the drainage afforded by the tube precludes the possibility of the development of toxic symptoms. Headaches may disappear following the use of purgatives, one or two hours before the bowel evacuation takes place. This is probably due to the advancement of the toxic material to the lower less absorptive areas, and would indicate that headache and other symptoms are attributable to intoxication rather than to the mechanics of the situation.

The blood-picture is that of simple secondary anemia and in the absence of acute symptoms, including fever, the polymorphonuclears are decreased and the small lymphocytes are increased. This change in the differential count is probably not the product resulting from colonic stasis, but is seen more especially in those having tooth and tonsillar infection coincidentally. Indeed, I have often felt that the colonic stasis itself is often a product of the debilitating influence of subinfection. Occasionally there is an increase of 6 to 10 per cent.

in eosinophils. That this is often caused by intestinal stasis is indicated by its reduction to normal with a reduction of the indicanuria and a return to normal colon function. Mild joint symptoms primarily induced are frequently encountered, or superimposed degrees of aggravation implanted upon already existing joint conditions are seen with sufficient frequency to justify colon investigation and treatment. Low-grade infections elsewhere in the body are aggravated by it, notably the post-pharyngeal lymphoid tissue and chronically infected accessory nasal sinuses. The liver falls heir to its parcel of bad influence of this intoxication and in time has its function definitely impaired. The chain of symptoms attributed by the older clinicians to biliousness is frequently of this origin. The patient, who is an abstainer, presenting the after-symptoms of an alcoholic debauche often is found by the test here presented to be laboring under a colon intoxication.

These individuals often bear the arsphenamins very badly. Any patient exhibiting unusual gastro-intestinal symptoms following the use of these drugs should be investigated from this standpoint, and if the urine be found to be heavily laden with indican the arsenicals should not be used again, else serious permanent liver damage might be inflicted. I do not use the arsenicals on those having a high indican content in the urine.

The vicious cycle so frequently commented upon in this connection had best be broken by instituting measures which will yield a perfect "score." The error should not be committed of discontinuing treatment directed to the detoxication if the symptoms do not disappear promptly. So long as large quantities of indican exist, conscience can be had to continue the treatment until it is no longer to be found, and even then the effect should be maintained by continuation of the management until the colon has been brought to function normally by virtue of a restoration of normal tone, and possibly also of normal position.

A disagreeable sensation of fulness and occlusion of the superior straits of the nose, occasionally but not always accompanied by mild asthmatic symptoms, has been seen associated

with colon stasis, relief of which caused this symptom to disappear.

In petit mal and grand mal, no more is claimed for the treatment than that many are benefited and some cured, when these cases are watched and treated until the urine is indican free. I would cite the instance of a little boy with petit mal, the attacks having been as frequent as 40 to 60 a day, who is now under treatment. Within four months the attacks have been reduced to 5 to 10 a day. Sunday morning headache, a distinct entity, depends for its unfailing periodicity on the delay of defecation occasioned by the late hour of rising which many enjoy on this day of rest. Breakfast is delayed and the morning evacuation may not be had.

The Chvostek sign, occasionally, and the Trousseau sign, less frequently, are seen accompanying high degrees of colon stasis and seem in no way connected with parathyroid tetany and its associated hypocalcemia. Those exhibiting these signs in this relationship frequently complain of "extreme nervousness" and an internal abdominal tremor, subjective in character.

Colon bacillus infections of the skin, tongue, ureter, and colon mucosa are apt to yield only indifferent results with any treatment when colon stasis is present, but when this is corrected cure often takes place spontaneously, and such supplementary treatment as vaccine therapy is attended with more brilliant results. It can undoubtedly be said safely that all cases of dysmenorrhea not dependent upon anatomic causes can be relieved, to a worthwhile or total degree, by employing the indican index in the use of an adequate régime directed to the usually existing colon stasis. I believe there are very few cases of dysmenorrhea, not in the anatomic group, that do not show high degrees of colon stasis. Here again, the test gives the physician the intelligence to carry on the treatment even though the symptoms may be slow to disappear, for the indican may be just as slow in effecting its disappearance as the symptoms.

The writer does not feel competent to speak with authority upon the relation of colon intoxication to internal secretion, but

it has been noted that there is a frequent relationship between hypothyroidism and this condition, and that the function of the thyroid is considerably enhanced by the restoration of normal colon and liver function. As accessory to the small low enema, either used alone or with drugs, I have found low abdominal support to be indispensable in many instances, especially in lean patients having the scaphoid abdomen. The Saur ptosis brace, made by Henry Saur of Philadelphia, has given the best results of any apparatus I have used; it is not so applicable to fat individuals. I would like to mention my preference for bismuth subnitrate as an intestinal antiseptic, using a teaspoonful dose at bed time twice a week for possibly two months at a time. As an admonition which has forced itself upon me, let me men-

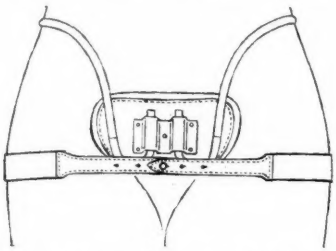

Fig. 274.—Saur's dropped stomach and colon supporter.

tion the use of laxatives in these sufferers having ileocecal incontinence in whom their use is accompanied by regurgitation, into the ileum, of cecal contents, with the attendant sudden onset of severe illness. Many of the individuals with whom calomel, castor oil, or the salines do not agree probably belong in this class.

In conclusion, I would urge that the plan of serially collected specimens of urine be used when indican is being looked for, and that when looked for in this manner it is a reliable guide in diagnosis and treatment.

In periodic health examinations this test would no doubt detect many individuals in the very beginning of colon stasis. There are many who have acquired the condition, in the stress of circumstances connected with busy lives, by unwittingly

making of the important business of defecation a perfunctory function. Often no quantitative significance is attached to it, and for this reason alone retention, intoxication, vicious cycle, and bad health results. Children are none the less liable, and with them the test is as applicable as with adults. Many times

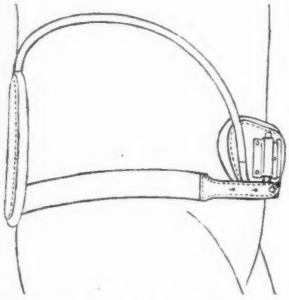

Fig. 275.—Saur's dropped stomach and colon supporter. Side view.

colons tasis in children is accompanied by a type of cyclic vomiting associated with acetonuria and a mistaken diagnosis of ketonemia is made. This and similar mistakes are unpardonable when a urinary test having such ease of performance is available.

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THE MEDICAL CLINICS OF NORTH AMERICA

VOLUME 10
1926 — 1927

PHILADELPHIA AND LONDON
W. B. SAUNDERS COMPANY

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PUBLISHED BI-MONTHLY (SIX NUMBERS A YEAR), BY W. B. SAUNDERS COMPANY, WEST WASHINGTON SQUARE, PHILADELPHIA.

MADE IN U. S. A

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